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GERIATRICS

THE

DISEASES OF OLD AGE AND THEIR TREATMENT

INCLUDING PHYSIOLOGICAL OLD AGE, HOME AND INSTITUTIONAL CARE, AND MEDICO-LEGAL RELATIONS

BY

I. L. NASCHER, M. D.

NEW YORK

With an Introduction by

A. JACOBI, M. D.

WITH 50 PLATES
CONTAINING 81 ILLUSTRATIONS

PHILADELPHIA
P. BLAKISTON'S SON & CO.
1012 WALNUT STREET

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PREFACE

No American work on senile diseases has appeared in over thirty years, the last being Charcot and Loomis' "Diseases of Old Age" published in 1881. Even that was not distinctively American for it was a translation of the published lectures delivered in the sixties by the great French physician in La Salpetriere, the home for the aged in Paris, to which were added ten lectures by Doctor A. L. Loomis of New York. Since then a lengthy article by Doctor A. Seidel of Berlin appeared in Wood's Monographs for March, 1890, and there have been a number of journal articles on various senile conditions, but no American work dealing with the subject as a whole has ever been published. The neglect of senile diseases (except arteriosclerosis which has received some attention in recent years) is evident from the paucity of literature on the subject. The cause of this neglect must be sought in the general mental attitude toward the aged. The spirit of veneration of ancestors and the aged, such as exists in China, does not exist among us. The sentimental interest in the aged is confined to the immediate family of the individual and there the interest is often less sentimental than dutiful. We realize that for all practical purposes the lives of the aged are useless, that they are often a burden to themselves, their family and to the community at large. Their appearance is generally unesthetic, their actions objectionable, their very existence often an incubus to those who in a spirit of humanity or duty take upon themselves the care of the aged. Those who would deny that this is the usual attitude toward the aged need but compare the treatment of the uncared-for child with the treatment of the uncared-for old man, the asylums for children with the asylums for the aged, the treatment in the home where children and their grandparents entail burdens upon the family. The physician views the aged from a different standpoint. As a humanitarian it is his duty to prolong life as long as there is life and to relieve distress wherever he may find it. There is, however, a natural reluctance to exert

oneself for those who are economically worthless and must remain so, or to strive against the inevitable, though there be the possibility of momentary success, or to devote time and effort in so unfruitful a field when both can be used to greater material advantage in other fields of medicine. Still these objections are paltry when applied to the physician's self-imposed obligation to relieve distress and prolong life. There is another point of view from which the physician should consider the aged and their diseases, that of the scientist, for here is a most interesting study, presenting problems that are intimately bound up in the grand mystery of life and death. In this direction the French and German investigators are far ahead of their American confrères, not so much in the quality of work done and positive results achieved, as in the quantity, the number of investigators, the many lines of investigation, and the opportunity afforded them to carry on scientific research. There the State takes an interest in scientific work, lending its aid, and there is substantial recognition of work accomplished. The lack of opportunity to carry on research work in this country except at a heavy expense to the individual, or else at the sacrifice of the credit and benefit arising from successful research work, is probably the main reason for the neglect of the scientific study of senility and its diseases. In recent years considerable work has been done in blood-pressure investigations, cancer research, arteriosclerosis and other factors related to senility and its diseases. The extent and depth of these investigations, which are really studies into the causes and results of senile changes, and the ever-increasing scope of these investigations, give promise of ultimate success in discovering the fundamental causes of senescence. Perhaps there may be controllable causes or causes which can be minimized so as to defer senility and prolong life to its physiological end. The prolongation of life is after all the aim and goal of the physician's endeavors. The author acknowledges his deficiencies both as a writer and investigator. Much of the histological and pathological data have been culled from other recent works, mainly German and French, but their erudite theories have been omitted except where theoretical discussions were necessary as in the chapter on causes of ageing. In nomenclature, the author follows the tendency of American writers to use English terms rather than the more scientific but often

more complex and less understood Latin terms used by Europeans. A few new terms are introduced where the old terms are complicated as "Grawitz' cachexia" to cover the disease he described under the name "fatal cachexia without discernible anatomical cause;" "Ortner's syndrome" for the disease he described as "dyspragia intermittens angiosclerotica intestinalis." The word senile is prefixed to diseases that present different features in senility from those of earlier life. Other terms like hypostatic edema, psychic senile debility, etc., will be readily understood. The classification is new. Lengthy descriptions have been avoided, minute pathology has been generally omitted, and only the essential symptoms necessary to recognize a disease and differentiate it from others have been introduced. It seems superfluous to describe every symptom that may appear in a disease in old age, which differs in but a few essentials from the similar disease of maturity, especially when the physician should know the disease as it appears in maturity or can get a description from the ordinary text-books. Greater stress has been laid upon the treatment of diseases and the differential diagnosis between normal senile conditions and pathological conditions which they simulate, as this branch of geriatrics has been generally neglected.

In presenting this work to the medical profession the author hopes to arouse an interest in geriatrics and stimulate research into the causes of senescence and the pathology of senile diseases. It is not too much to expect that as a result of such interest and research we will get a better knowledge of the senile organism and be more successful in coping with senile diseases than we are at present. Believing that attention would be more readily concentrated upon this subject if it were considered entirely apart from maturity, the author suggested that it be studied as a special branch of medicine to which he applied the term *geriatrics*. This term which has been generally adopted is derived from the Greek, *geron*, old man, and *iatrikos*, medical treatment. The etymological construction is faulty but euphony and mnemonic expediency were considered of more importance than correct grammatical construction.

I desire to express my gratitude to the authors and radiographers who have furnished me with cuts and illustrations and the permission to use their illustrations which appear in this

work; also to the Journal of the American Medical Association, Medical Record, and New York Medical Journal, in which some of these illustrations first appeared; and to Doctor Alexander Klein of Philadelphia for his careful revision of the entire work.

I would also like to express my gratitude to Doctor Robert Abrahams and the Medical Staff of the Home of the Daughters of Jacob, New York, and to Doctor W. Travis Gibb and the Medical Board of the New York City Home for the Aged and Infirm, and the General and Neurological Hospital for the opportunities given me to study cases at those institutions.

Some of the material in this work appeared in the author's papers which were published in the N. Y. Medical Journal, Medical Record, Medical Times, American Medicine, American Practitioner, Archives of Diagnosis, Dietetic and Hygienic Gazette, all of New York; International Clinics and Medical Council of Philadelphia; and Am. Journal of Clinical Medicine of Chicago.

I. L. Nascher.

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INTRODUCTION

The physiology, pathology and therapy of early age have been extensively studied and discussed in our country these fifty years. Whatever American contributions to pediatrics there were before 1800, could easily be enumerated on a few pages of "Janus, 1900." After that time there were a few books by Dewees, Eberle, Stewart, Condie and Meigs, but the interest in pediatrics of our medical profession was not an animated one until half a century ago. That was for Europe the time of the epochmakers Rilliet and Barthez, Charles West, and Karl Gerhardt. With us pediatric literature and the taste for it, and the appreciation of its necessity and dignity have deservedly grown since. Magazine articles, laboratory and bedside reports, pamphlets and comprehensive books—some say too many—have increased to such an extent as to form a voluminous library. Possibly, however, not all of these works, mainly the deluge of text books on the diseases of children, are indispensable, many may have been merely the offsprings of the author's autosuggestions. Still, even they demonstrate the force of the new movement and the extent of the new market. A few special journals have also proven the growing interest in pedology which was exhibited both by the profession and the teachers who are mostly guided by the suggestions and demands of the medical public as represented in our large city, state, and national associations. This tendency, however, has fortunately not succeeded in building up a new specialty with all its narrowing influences; on the contrary, it has broadened the horizon of honest students and made better general practitioners out of those whose main endeavors were diverted to understanding all about the young. At the same time those whose interests were originally confined to the study of and practice among the people at large, added to their intellectual capital by acquiring the results of specialistic labors in pediatrics. I am quite sure that the pediatrist cannot succeed in his practical work without being a close student of nosology in general, and on the

other hand, that the "internist," the general practitioner and physician does not, without being at the same time a pediatrist, reach a standard by which he may be of real use either at the individual bedside, or as a sanitarian in the councils of the nation. The world is entitled to demand of every one of us a complete knowledge of and profound interest in the physical life both of the young and the old.

Now why is it that the growing interests in many of the branches of medical science and practice has not equally been extended to the diseases of old age? It might properly be measured by the literature of the subject; but it has not manifested itself by books or pamphlets or magazines, nor by a specialistic instruction in our American schools of medicine. Nor has any of our great and influential associations insisted upon the appropriate enlargement of the medical curricula of our teaching institutions. Our American literature is poverty stricken, comparatively. For the brilliant labors of C. S. Minot in part published in the Popular Science Monthly, with his "Problem of age, growth and death" (London, 1908) has not yet fertilized our desert. The British literature to which we should resort, is not much richer. Day, 1849, Mclaghlan, 1863, G. M. Humphrey, 1885, G. W. Balfour, 1894, Clifford Albutt, 1896 are the only books which have treated of old age, monographically. A few translations, mainly that of Charcot's lectures on senile diseases contained in the new Sydenham society's publications (vol. 95) 1881 furnish the best there was of what now-a-days is considered old literature. Finally the eternally young Sir Herman Weber has given us in two editions of "on means for the prolongation of life" in his best style his philosophical and clinical views on how to remain young when advanced in years, and Robert Soundby acknowledging the defects of the literature of his country, has published a very competent clinical guide ("Old age, its care and treatment in health and disease," London, 1913).

France has not been very productive. Still, after Gillette, 1851, and Reveillé-Paris, 1853, Charcot's original work was a great achievement, rich and fertile. He was followed by Demange in 1886. Boy-Teissier's lectures of 1895 and G. Rauzier's book of 1909 are valuable and influential works. The latter seem to have diverted the attention of young

authors to the subject which was rather neglected; indeed during the last few years more than a dozen Paris inaugural theses have been published; they treat monographically of old or senile organs.

Germany has proven its supremacy as the modern leader in medical science through its contributions to what Doctor Nascher proposes to teach under the heading of geriatrics. After Fischer's Tractatus de senio, 1766, comprehensive treatises have been furnished by Canstatt, 1839; Geist, 1860; Mettenheimer, 1863; F. W. Müller, 1863; Seidel, 1889; Mühlmann, 1900; F. Friedmann, 1902; Schwalbe, 1909 Lindheim, 1909 and Arne Faber, 1912. A vast number of German monographs, clinical, pathological, histological and therapeutical, have added to our knowledge. Aschoff's many studies published during the last few years will long be our guides in the appreciation of the dignity and import of the advancing changes of the blood-vessels.

Dr. Nascher has undertaken to write, what for our country seems to be the first modern comprehensive book on the normal and the morbid changes of old age. He has honored me by permitting me to accompany it with this introduction to the medical public. This work has been suggested to him by his scientific interest, continued study and humane sympathy. He does not mean to take the sufferings of old age and early death for granted, and for welcome dispensations of providence. That may be the point of view sufficient for the statistician, while the individual, beyond the threatened premature decay, has a justifiable claim to comparative health, persistent comfort, and uninterrupted efficiency. These are the great assets of the individual who looks for competency and enjoyment, and of the human society which has a right to demand cooperative services from all. For premature incompetency and premature death mean private and collective bereavement. It is the domain of the physician for whom this book is written to combat them. If it be correct that sclerosis and atheromatosis and cell atrophy and malignant proliferations are natural results of histological changes resulting in vital retrogression or malignant degeneration, and in sufferings and dangers, they can and should be delayed and rendered less formidable or even innocuous by the very props and staffs of childhood and adult life, viz., hygiene, diet, drugs and surgical aids.

The study of advanced age will enhance the competency of the physician to the same degree to which it was advanced by the closer knowledge of the physiology and pathology of the infant and child. With this difference: the baby offers but few difficulties in arriving at a diagnosis. His diseases are simple. He has only one at a time. Complications are infrequent, but the perplexities grow from decade to decade. For there are only few diseases that leave no remnants. The recovery from every new disease contracted at any period of life is handicapped by the tissue changes left behind from previous accidents or ailments. There are few persons of advanced years without a permanent blemish—one or many—which make the diagnosis of any additional illness or morbid condition more difficult, treatment more uncertain, and complete recovery more doubtful. That is why I imagine that Doctor Nascher by offering the practitioner of medicine this book, will render him a meritorious service.

A Jacobi

GERIATRICS

CHILDHOOD AND OLD AGE

Senility is often called *Second Childhood.* A comparison of the organism in childhood with the organism in old age will show that there is not an organ or tissue, not a function, mental or physical, identical at the two periods of life. Vitality, metabolism, even instinct differ. The process of senescence is progressive, not retrogressive, there is no reversal in the order of development and not a single tissue reverts to an earlier type.

If we accept the theory of tissue cell evolution as the fundamental cause of ageing, we must seek the fundamental difference between childhood and old age in the cells at the two periods of life. There are however profound differences in the organs as entities and in the organism as a whole. While the gross differences are obvious or demonstrable, we have but slight knowledge of the changes in the cell. It is probable, however, that some of the cell and tissue changes are not inherent but are caused by some change in nutrition. We have not yet discovered any change in the blood at the two periods except in the proportion of salts and in viscosity, although the spleen and bone marrow are greatly altered in advanced age.

It would carry us beyond the scope of this work to discuss the kinship between chemical and physical affinities such as occur in simple substances like potassium for oxygen and its oxide for water, and the elective affinities of complex substances like protoplasm for the complex substances they require as pabulum. As a result of the fulfilment of the elective affinities in the organism there is going on a constant change of chemical combination, cyclic, uniform, unchanging in character, the like pabulum constituents being converted into like substances forming body or waste. Neither chemist, cytologist nor physiologist has been able to explain the biochemical changes in the cell or the metabolic changes in the organism or demonstrate order in them by formula or law. Any school boy can show by sym-

bols how amorphous phosphorus will combine with calcium and oxygen to form the tribasic phosphate of calcium, but no scientist has yet explained how this combination is brought about in the body since this form of phosphorus is insoluble, even in serum and the tricalcic phosphate, in which form it is eliminated, is insoluble in water and but slightly soluble in weak acids. Shall we say that living blood has solvent powers not possessed by any other solvent? How can we explain the normally increased retention of lime in the aged and its deposit in locations in which it is never found in early life, except in disease? We must assume that the early cells, which show intense greed for pabulum, will not take up more lime than the organism requires for healthy growth and they may take up less, causing rickets and similar conditions of lime deficiency, while the aged cells, in spite of their lessened appetite, show a greater elective affinity for lime. We must also assume that the blood has a greater affinity for lime while the metabolic processes are so altered that less is eliminated. Minot has shown that there is an increase in protoplasm in aged cells but this alone would hardly explain the profound differences in cell activities at different periods of life. It is probable that there is a difference in the character of the protoplasm itself and perhaps in the nuclear constituents, since recent investigations have demonstrated dissimilarities in the chemical composition of the proteids of different cells, which were supposed to be identical. Greater refinement in chemical analysis and increased microscopic power will undoubtedly reveal chemical differences and organic changes which will clear up these problems.

At the present moment there is but one rational assumption by which we can explain the progressive changes in the properties of cells and the tissues which they form. It is, that in the constant waste and repair of tissue the newer cells differ from the earlier ones, that in advanced life none of the early cells are left (except brain cells), that the aged individual is in fact an entirely different individual from the one who was formed from the ancestors of the late cells. The only connecting link between the child organism and the senile organism is the brain, as it is believed that brain cells do not regenerate themselves, that the old cells were all present at birth though changed in structure and perhaps in composition in the process of development and

senescence. There is still the same personality, modified by intelligence, education and the acquisition and suppression of traits. Continuity of activity is maintained by retention of sentience in the original cells, instead of by transmission from generation to generation of cells as in other tissues. Like the old vessel which has been repeatedly repaired until not a splinter of the original timbers is left, the individuality and the name remain.

Growth in youth depends primarily upon nutrition. The underfed child is also underdeveloped and no amount of overfeeding after the developmental period will increase the growth of undeveloped tissues. When well fed children are underdeveloped there is usually a dyscrasia causing impaired general metabolism, or there may be deficient digestion and assimilation or else general cell sluggishness, usually a transmitted quality. Whatever the cause may be the whole physical organism suffers but mentality is rarely impaired. In the atrophy of advanced life there is no uniformity in cause, extent or mode of procedure and like tissues may undergo different forms of degeneration in different parts of the body. Some organs and tissues degenerate earlier and more rapidly than others but with few exceptions, as the thymus gland and the female generative organs, there is no time or regularity in the order of the senile degenerations. Inactive striped muscular fiber degenerates early and undergoes fatty infiltration and degeneration. Active striped muscular fiber does not degenerate until it has reached its maximum growth after which the extent of degeneration depends upon the activity or work it is called upon to perform. If it is not excessively employed it degenerates late and then atrophies with loss of power proportionate to the waste of tissue. If excessively employed, there is loss of tonicity and a change in the character of the fiber, usually a fatty degeneration. When healthy tissue normally employed atrophies we look for a nutritional fault and we generally find an impaired blood-supply. In old age we have altered cells and supposedly altered blood. Do these aged cells require a different pabulum from the earlier cells? Does the blood in the aged carry insufficient cell nutriment or are the nutritional constituents so changed as to be unsuitable, or does it carry constituents inimical to cell life? Transfusion of the blood of a young person into an old person apparently does not inhibit

senile changes nor does the blood of an old individual into a younger one induce such changes. Further experimentation along these lines is necessary to determine the influence of the blood at different ages upon young and old cells. (W. T. Gibb suggested to the author the transfusion of blood from an old member of a family possessing hereditary longevity into a young member of a short lived family for the purpose of promoting longevity.)

There are however other causes for senile tissue atrophy than the fundamental changes in the cells and the probable change in the blood. Connective-tissue proliferation may compress tissue cells as in the liver, bands of connective tissue may compress blood-vessels and lessen the blood-supply as occurs in the spleen, or the swelling of endothelial cells may diminish the caliber of vessels as occurs in the vasa vasorum.

We have still to consider the differences in vitality, metabolism and mentality in the two extremes of life. Under vitality will be included irritability or the property of responding to external stimuli, sentience or automatism independent of external stimulus, vital energy and vital resistance. (The term sentience is used here to designate the property of originating action independent of irritation or purpose. This would include instinctive acts and acts performed unconsciously though such are not usually included in this term.) While these properties are intimately related they will be dealt with separately.

Irritability is pronounced in childhood and weakened in old age. It requires a much greater stimulus to the aged sense organs to rouse sense perception and the responses are slower than in earlier life, and the same applies to tissues where the senses are not involved. The ciliated epithelium for example is much more sensitive in the child and a slight irritation to the cells is followed by stimulation of the glands in the underlying mucous membrane. For this reason the mucus expectorated by the child is usually clear while the mucus expectorated by the aged individual is usually dark from dust particles which had accumulated on the membrane without causing enough irritation to the ciliated epithelium to induce immediate coughing to dislodge them. Reflex action in the young follows initial irritation rapidly and instinctive acts are readily aroused upon slight stimulus while in the aged reflex action is slowed and weakened and instinctive

acts are rare. Evidences of diminished irritability in the aged are obvious in almost every act they perform. We must remember however that slowed responses may also be due to weakened mentality, a longer time being required to translate the irritation and determine the response.

Sentience is active in the young, weak in the aged. The regulating centers in the aged are weakened and while some are easily disturbed others require a powerful stimulus to cause any change in their activity. The activity of the heat regulating center is lessened and there is a general lower temperature with a normal range of about two degrees in the course of the day. In old age some profound influence is necessary to raise the temperature three degrees while in childhood with a normal range of a degree or less, slight influences will stimulate this center and cause a rapid rise of several degrees with an equally rapid fall to normal. The heart-regulating centers are easily disturbed in the aged, the respiratory center can stand but little disturbance while the vasomotor center is in a constant state of unstable activity. In the young functional disturbances not due to anatomic changes are quickly regulated and normal functions are restored without serious impairment of the organs; aged tissues cannot readily accommodate themselves to functional changes and they quickly degenerate. Automatic activity such as respiration, heart action, peristalsis, glandular action, and voluntary sentient acts as deglutition without food irritation, the control of the sphincters, the swinging of the arms when walking, are all performed less energetically in old age. Early life is marked by cellular activity; age, by cell sluggishness.

Vital energy gradually diminishes with age except during the menopause, critical period of the male and the senile climacteric. These periods are marked by increased mental, physical and metabolic activity and are followed by rapidly diminishing activity and energy. In youth there is a wide margin between normal functional activity and the limit of functional capacity. In advanced age the normal functional activity is diminished but the limit of functional capacity is lowered much faster and the margin between the two is gradually lessened. Activity is maintained by vital energy, but when carried to the limit of functional capacity, further activity causes exhaustion or paralysis or, in the case of blood-vessels, rupture. In the young person,

after running, the heart will beat faster, respiration is more rapid and all the organs and tissues show the effect of greatly accelerated circulation. The young person is forced to stop through muscle exhaustion, complete recuperation following rest. Only in case of heart disease is there any danger from heart exhaustion. In the aged the limit of functional capacity is reached before muscle exhaustion sets in and death may occur from heart exhaustion, respiratory paralysis or rupture of an atheromatous artery. This does not show diminished vital energy but diminished functional capacity. Diminished vital energy is shown by the greater effort or impulse required to perform acts. Acts now require a sensible effort and a conscious purpose which were formerly performed unconsciously or without any conscious mental or physical effort, as the regulation of the step when walking or the arm movement when conveying food to the mouth, swallowing, recalling a familiar name or simple relations between things, listening, seeing, crossing the legs, etc. The child in play runs to hide. The impulse to run is sudden and instantaneous and no thought is given to the movement of the legs in the act of running. The energy expended is so slight as to be unnoticed unless fatigue, palpitation or dyspnea sets in. The old man needs a conscious impulse, a mental push, to start running and his thoughts are on the act instead of its purpose. He may walk absent mindedly as this requires little effort or energy but he will not run absent mindedly.

Vital energy is sometimes divided into three forces, bathmism or growth force, neurism or nerve-force and phrenism or brain force. In childhood the growth force is exerted in two directions, or rather with two distinct purposes, accumulation of tissue and differentiation of the sexes. In old age there is still growth of tissue but the new tissue does not fully compensate for the waste except in a few tissues. This growth force is now mainly exerted toward the approximation of the sexes and in old age they approach a neutral type. This is more pronounced in the virilescence of the female. In the female there is usually a growth of hair upon the face, while the hair on the face of the male becomes thin. Her voice becomes lower, his becomes higher in pitch. The changes in the male pelvis and in the neck of the femur produce a greater width between the crests of the

ilia, and the proportion between width at the hips and length of the spinal column is greater in the aged man than in the younger male and may equal the proportion found in the female. The pelvis of the female infant is of the male type while the pelvis of the aged male approaches the female type. The thoracic changes are the same in both sexes and in the female the breasts shrivel. The changes in the lower maxilla in advanced age give to both the weazened face and there is often the same facial expression. We frequently see photographs of aged individuals in which the face alone gives as little indication of the sex as the face of the infant. The diminution in nerve-force needs no discussion as it is evident in every act of the aged individual. The alteration in brain force will be taken up under mentality.

Vital resistance or the opposition of the living organism to deleterious influences differs at the two periods of life. The young are much more susceptible to infectious diseases than the aged and the eruptive diseases of early life rarely or never occur in the aged while other bacterial diseases occurring in the aged are milder. Various explanations have been given for this phenomenon, such as poor soil, lower temperature, more opsonins, more active leucocytosis, etc. We are again confronted by the question, is the blood of the aged essentially different from the blood of the child? Is there any difference in the character and activity of the cells?

The child can stand changes in temperature, atmospheric pressure, environment and mode of life better than the aged. Dietary changes affect the child more powerfully. While the child is readily affected by deleterious influences and inflammatory conditions are easily produced, the young organism can accommodate itself to such influences; and if disease occurs vital energy maintains functional activity until the organs or tissues involved are restored to their normal condition. In the aged inflammatory conditions are infrequent, when disease occurs the healthy senile organs cannot readily accommodate themselves to functional changes in diseased organs, the functions are maintained with difficulty owing to diminished vital energy and little or no reserve energy, and tissues and their functions remain impaired or very slowly recover to their normal *senile* state.

Metabolic activity is altered in old age and markedly

different from metabolic activity in childhood. In childhood there is active destructive and constructive metabolism, the regeneration being in excess of the waste. Stöhr says "a femur of a three year old child contains scarcely any of the osseous tissue present at birth." In old age metabolic activity is lessened, the anabolic processes being less active than the catabolic processes. Insufficient repair is found more especially in the higher order of tissues, the brain, marrow, spleen and muscle, tissues which require a plentiful supply of blood for their nutrition, while in the lower order of tissues like connective tissue, fat and hair there may be increased growth. Assimilation is altered in advanced age, many substances which in earlier life are retained and converted being now rejected and thrown out in the feces. The intestinal decomposition products are increased, the total amount of urea and uric acid eliminated is greatly diminished, only about half of the amount of CO_2 exhaled in early maturity is given off in senility, while the elimination of waste by the skin is very small. Abnormal fatty acids are produced and eliminated as fetid perspiration. Indican is always present in the urine. A smaller amount of food is required by the system in old age and the excess of food is thrown off in a lienteric diarrhea. The water content of the blood and tissues is diminished but increased liquid ingesta increases the urine output without relieving the dryness of the tissues or diminishing the viscosity of the blood. Salines which increase the fluidity of the blood are readily absorbed but are rapidly eliminated, while calcium salts which increase the viscosity of the blood are retained. The natural adaptation of tastes, wants and supply to the needs of the organism is beautifully illustrated in the aged. There is diminished activity and lessened need for carbohydrates and there is a distaste for sweets. The bile is diminished and there is a distaste for fats. With the falling out of the teeth there is a dislike for meat, which must be masticated. The amount of hydrochloric acid in the stomach is diminished and the aged individual craves for sour and salty things while insipid foods, which are usually alkaline, are rejected. If, through the trickery of the cook or the perversion of taste, inappropriate or excessive food is taken, it is eliminated by the bowels unchanged or but slightly converted. The child usually vomits inappropriate food but food

in excess is retained and stored. This is especially marked when sweets are taken in excess, and accounts for the chubby figures of children and young women in places where much sweets are used. The fancied similarity in the mentality of childhood and old age gives rise to the belief that senility is second childhood. Only in the complete absence of intelligence of the newborn infant and the absolute dement is there any resemblance in their mentality. Even then the child performs instinctive acts and gives evidence of sensations as pain, hunger and disagreeable impressions which are absent in the complete dement. The child is guided by ancestral knowledge or instinct but such knowledge has virtually disappeared in old age. Whatever acts the aged individual performs are the result of a conscious purpose and reason or else of habit or irritation. Sense perception is strong in childhood, weak in old age. This weakness is due partly to impairment of the sense organs, partly to weakened mental perception. Memory is strongly developed in the child; it receives impressions, stores them and recalls them at will as mental pictures, sounds or other sensations, without apparent effort. In the aged only powerful impressions or those directly affecting the individual are retained. A sensible effort must be made to recall earlier impressions although very early impressions will reappear without effort or design and the aged person boasts of his wonderful memory. Reason is a late acquisition of the child and persists late in the aged. The child's mind is analytical; it wants to know *why*, and it will take apart, destroy, question. The senile mind is synthetical; it wants to know *how*, to combine, to construct and to restore. In rare cases children will construct and aged persons will analyze and destroy. We call these geniuses. In other cases individuals will perform remarkable constructive work at an advanced age. Here we will usually find all mental efforts directed into one channel and the particular work stands out prominently while in every other direction the mental faculties are deficient. There are differences in judgment, imagination, the ethical sense, the esthetic sense, sentiment and other mental traits and characteristics between childhood and old age but these differ so widely in individuals of the same age that we cannot make a broad distinction at the two periods of life. The same applies to the will, although the aged generally will follow the lines of least

resistance, become subjective and submit choice and resolution to the will of others. The child in its general conception of life and the world gives no thought to its somatic self; the aged gradually constricts his conception of life and the world until it is centered upon himself; his interests are all concentrated in the preservation of his life. While the fundamental difference between the young and the old organism must be sought in some essential change in the character of the cell, the fundamental difference between childhood and old age can be summed up in this. *Youth wants to know; age wants to be.*

PART I

PHYSIOLOGICAL OLD AGE

THE SENILE STATE

We cannot deal understandingly with senile diseases if we do not understand the *senile organism*. We cannot understand the *senile organism* unless we study it as a physiological entity entirely apart from maturity. The physician must look upon old age as he does upon childhood. His conception of the child is not of an adult with undeveloped organs and tissues, nor does he deal with the diseases of that period of life as though they were diseases of maturity complicated with immature development. A pulse of 120 in an infant does not mean tachycardia nor does limited reasoning power stamp the infant as an idiot. These conditions are natural and normal at that period of life although they are unnatural, abnormal and pathological in maturity. We must take a similar view of senility. We must look upon the degenerations, the atrophies, hypertrophies and all the changes in form and character, that are due to the process of involution, as natural, normal and physiological. The brittleness of bone in the aged, due to the waste of organic matter and the proportionate excess of lime salts, is as natural as is the softness and elasticity of bone in childhood when there is still an insufficiency of lime salts. Senile debility is no more a pathological condition than is the weakness of the infant, senile contracted kidney is not Bright's disease although it resembles interstitial nephritis; the hardened, contracted capsular ligament is not a disease of metabolism although the stiffness occasioned thereby and the pain on motion resemble rheumatism. The irregularity in the order and the wide variations in time and extent of the senile changes in different individuals make it impossible to establish a norm or standard for these changes. Neither can we determine the extent of the senile process of involution, from the individual. It is not unusual to find an individual presenting the appearance of extreme decrepitude without marked changes in the internal

organs and, on the other hand, we sometimes find apparently robust individuals with early signs of arteriosclerosis, cardiac hypertrophy, and the whole train of changes that arise from defective nutrition and elimination of waste, following senile changes in the circulatory system. Another difficulty in the way of determining a norm or standard of senile types is the impossibility of fixing averages such as serve for determining standards in maturity. In maturity the anatomical condition and the physiological function generally bear a definite relation to each other. In advanced age we frequently find degenerative changes without marked noticeable change in function; indeed, we may find the changes due to age occur in early maturity while functional activity may increase. The brain reaches its maximum weight about the thirtieth year after which there is a gradual loss of weight, yet the maximum mental capacity is generally reached about the fiftieth year or later. The lungs reach the maximum respiratory capacity about the thirtieth year, and a diminution from this maximum capacity has been demonstrated before the fortieth year while the earliest symptom of impaired respiration, dyspnea, does not usually manifest itself before the middle of the sixth decade of life. Since we are unable under these circumstances to establish a standard based upon either age, or extent or character of morphological changes it will be necessary to use extreme types for the purpose of description. We must remember, however, that even such types may be normally exceeded, while under some circumstances slight deviations from the norm of maturity may be pathological. We also find occasionally a pathological condition which has existed for so long a time that it has become normal to the individual. Such cases will receive no further consideration.

The obvious characteristics of senility are evidenced in the appearance, attitude, gait, mentality and the tout ensemble of mental and physical decay. The appearance of the senile individual is repellent both to the esthetic sense and to the sense of independence, that sense or mental attitude that the human race holds toward the self-reliant and self-dependent. It is not within the scope of this work to discuss the psychonomy of the emotions; this much is however certain: While the dependence of the child arouses sympathy, in the aged the repugnance aroused by the disagreeable facial aspect and the

idea of economic worthlessness destroys the sympathy we bestow upon the child and instills a spirit of irritability if not positive enmity against the helplessness of the aged. We find herein one of the causes for the general neglect of the aged, where this spirit is not overcome by a spirit of reverence. The mental depression and the lack of interest in things beyond the ego of the aged individual contribute to the general feeling of repulsion and all these factors accentuate the disagreeable tout ensemble of old age. The countenance is either expressionless, indicating mental weakness, or there is an apathetic moroseness indicative of helpless resignation, or else there is the anxious look associated with a haunting fear. The skin is dry, lusterless, darker than in maturity, often pigmented, loose and thin, showing varicosed veins and tortuous arteries underneath. In some localities the skin lies in folds producing coarse and fine wrinkles. This is due partly to the looseness of the skin itself and partly to the waste of muscular fibers and fat tissue. The hair is thin, gray or white, there is often baldness, sometimes there is an excessive growth of hair in unusual places as in the nose, ears, eyebrows, and on the upper lip of women. The nails become brittle and are frequently cracked, they generally show neglect, the ends being broken or worn off. Owing to the impaired circulation and defective oxygenation of the blood there are usually cyanosed lips, pale ears and areas of passive hyperemia over the malars and at the tip of the nose. The waste of the muscles is determined by their activity. In actors and public speakers who make frequent use of the facial muscles in giving expression, these muscles waste in bulk, they present tense borders leaving the muscles in sharp outline. In these cases the muscle texture remains unchanged. Where the facial muscles have not been much employed they become subjected to fatty infiltration, the muscles waste late, they leave no sharp borders and they are soft and flabby. This condition is well seen in the dull ignorant peasant in whom the masseters show the waste due to activity while other facial muscles present the changes referred to. In this class too do we find the skin much darker owing to exposure and rough treatment.

A marked senile characteristic which itself gives the impression of lack of energy is the atrophy of the lower maxilla, producing the so-called weak chin of the physiognomists. This

atrophy includes loss of the teeth and waste of the alveolar process, a more obtuse angle of the jaw and changes in the articular surfaces, causing changes in the anatomical relations of the bones of the face. The eyes are generally lusterless and present a gray ring around the cornea, the arcus senilis. There is frequently a ptosis of the upper lids and occasionally a mild ectropion. The attitude of age is well described as a slouch. The stature is diminished through compression of the intervertebral discs, exaggeration of the spinal curvatures, flattening of the pelvis, depression of the neck of the femur and generally broken-down arches. There is also an apparent decrease in stature owing to the droop of the head and the bent knees, the former being due to weakness and waste of muscle, the latter being caused by the effort of the individual to maintain equilibrium. A psychic cause for this senile slouch will be described under senile debility. The senile gait, the "abasia senescentium" of Petrens, is a halting walk with slow, short, uncertain steps. Naunyn calls it a neurosis due to impaired coordination. I am inclined to ascribe this gait to a weakening of the subconscious control by which we regulate our walk, the weakness of the muscles, slowed motor impulses and the stiffening of the joints in senility. In addition there is usually some pedal defect such as broken-down arches, hammer toes, bunions, etc.

The most profound changes occur in the functions of the brain. The many complex factors embraced in the term mentality, the uncertainty of their interrelations and our ignorance of the mode of action of the brain preclude any lengthy discussion of this subject. The senile changes in mentality are found in temperament, emotions, will, sensations and intellect. The most prominent mental characteristic in old age is an overwhelming interest in self, a selfishness which gradually subordinates every other interest in life to the welfare of the individual. Notwithstanding all the optimistic platitudes of philosophers from the days of Cicero to Metchnikoff, notwithstanding the inbred resignation of the fatalists, the ready submission to the inevitable of the materialists, notwithstanding the promise of heaven, bliss and light and life everlasting, made by theologians of all ages, man looks forward to death with dread and indignation. And the nearer he approaches the abyss beyond which, he is told, lies eternal life, the greater his dread, the more pro-

found is his sense of impotence, the more depressing is his resignation. In the healthy mind of maturity thoughts of death, when they arise, are set aside, for future reference as it were, unless some circumstance momentarily forces attention to death. When, however, the infirmities of age bring such thoughts persistently and with ever-increasing intensity to the individual, life assumes a value incomprehensible to the younger mind. With increasing infirmities and the realization that the span of life is rapidly nearing its end, the desire to live becomes the all-absorbing thought. In this intense desire to live we find the basis of the selfishness of the aged. It is also the cause of his suspiciousness, his egoism and temperamental changes. There are contributing causes which may in some instances be more potent than the causes just stated. The fear of leaving a family unprovided for, the fear of becoming a burden to the family, or friends, or the State, may produce a moroseness and depression which would change the temperament of the individual. Likewise would the irritability caused by discomforts produce the same effect. These changes affect the emotions and, as the reasoning power diminishes, its ability to control the emotions wanes.

In old age a stubborn unreasoning perverseness often takes the place of a reasonable strong will. Of the intellectual faculties memory is usually the first to show impairment, names and numbers being quickly forgotten. Recent events unless directly affecting the individual are not firmly impressed upon the mind and are soon forgotten, while early events are readily recalled. In those accustomed to employ the reasoning faculty, this faculty generally remains unimpaired so far as the quality of the work is concerned, but greater mental effort is required and brain fatigue sets in more rapidly. In some individuals all of the intellectual faculties become uniformly weakened, producing a progressive senile dementia. There is a marked change in mentality during the senile climacteric which will be described further on.

In this brief review of the obvious changes that occur as a result of ageing, special stress has been laid upon the mental changes, as they are often the first indications that the period of decline has begun. Lessened interest in the events of the day, a tendency to sleep after some mental work, greater

difficulty in getting ideas or some particular word to express ideas, forgetfulness, all point to senile changes in the brain.

The subjective indications of advancing age do not correspond with the objective manifestations. In many cases of men the first change which attracts the attention of the individual is lessened sexual power without diminished desire. Occasionally the desire first wanes and in such cases there arises often sexual perversion. In many cases the individual complains of pains and aches in the muscles and joints which he ascribes to rheumatism, or of shortness of breath which he says is asthma, or of a desire to sleep after ordinary mental or physical work and this he calls malaria. Some men will take the first gray hair as an indication of ageing and this is the only obvious manifestation which the individual will notice before others. Many persons will deny any feeling of age, even when such pronounced symptoms as dyspnea, palpitation of the heart, pains and aches in muscles and joints, and diminished capacity for all kinds of work are present.

While nearly all that has been said applies to women as well as to men, there are some differences in both the objective and subjective manifestations between the sexes. Many women begin to lose energy and power immediately after the menopause, and this may be looked upon as the earliest of the subjective manifestations of ageing. Objectively we find a growth of hair upon the upper lip, a waste of the muscles of the neck, deposit of adipose tissue upon the abdomen. The senile kyphosis is not as marked in women as in men. This is due partly to the effort to maintain an erect bearing and to present a pleasing appearance, partly to the support given to the back by corsets and stays, and partly to the slighter downward pressure exerted upon the spinal column by wearing the dresses suspended from the hips instead of from the shoulders. The mental changes in the female generally include all the intellectual faculties and proceed to the extent of complete dementia far more often than in the male.

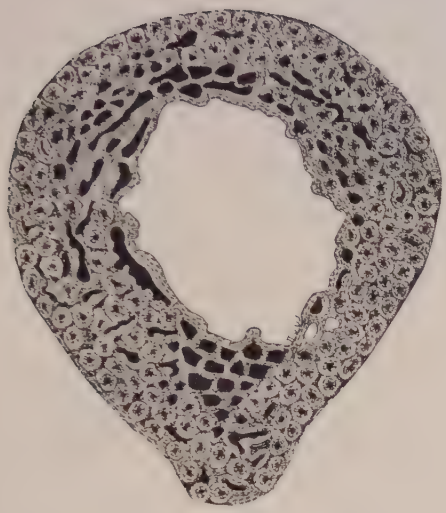
The obvious manifestations of senility appear later in the female, for the reason that she makes an effort to remain attractive, the psychic factor involved in the production of the senile slouch in the male being overcome by her vanity, there is absent the senile kyphosis, the marked waste of the facial

muscles, and often the wrinkles generally seen in the male. Women being more impressionable than men, they are more amenable to religious teachings, they become more readily resigned to the inevitable through their faith and hope of eternal life hereafter, and being more cheerful they do not present the disagreeable, gloomy appearance of aged men. This as well as their sex brings to them the sympathy denied to men.

Our conception of old age must be based upon the harmonization of the objective manifestations, of the subjective manifestations and the organic (physical and mental) changes so far as we can determine them. In considering the objective symptoms we must exclude the slouch due to laziness, the careworn expression due to worry, the waste of muscle from disease and insufficient food, the roughened skin due to exposure or improper treatment, the kyphosis due to certain vocations as well as to disease, the peculiar gait of various nervous disorders and the mental weakness of cerebral disease. The subjective symptoms may be due to various diseases. Of the organic changes only one has been found to be invariably due to ageing. This is the progressive increase of interstitial fibers between the pyramids of the kidneys, first described by Doctor Jos. Walsh of Philadelphia. Every other senile change in the organism may also be found as a pathological process in maturity, and it is often difficult to determine whether the change is due to ageing or to disease. The difficulty is increased by the fact that changes due to ageing have been demonstrated in early maturity yet give no manifestations, objective or subjective, until two or three decades later. Diminution in respiratory capacity begins about the end of the fourth decade yet difficult respiration due to the atrophy of the lungs may not manifest itself until the sixth decade or later. The brain begins to lose in weight during the fourth decade, sclerotic and atheromatous changes in the blood-vessels without apparent cause or complicating disease have been observed in the third decade, while cardiac hypertrophy has been found in athletes before the third decade. The popular conception of old age is based upon the appearance of the individual. It is not unusual, however, to find apparently decrepit individuals regain strength, mental activity, cheerfulness and a more buoyant spirit as well as a more youthful appearance when freed from care and the necessity to work.

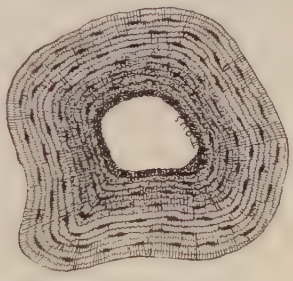
This is a common observation in inmates of homes for the aged, shortly after their admission. A conception of old age based upon the subjective manifestations may be equally fallacious, as these may be symptoms of true pathological processes, or due to temporary psychic influences. Neither can we base our conception of old age upon the organic changes due to ageing, as these may appear in early maturity. The term old age should be applied only to such cases as present obvious manifestations or marked subjective symptoms with the progressive organic changes which are due to ageing. The term senility is usually applied to a more advanced old age. It implies pronounced senile changes with the accompanying objective and subjective manifestations, and covers the period from the time when the mental and physical impairment begins to incapacitate the individual, to the complete decrepitude that ends in physiological death. It corresponds to the postclimacteric stage of the period of decline.

During this climacteric there is a readjustment in the relations between the functions, and changes in the organs necessary to carry out the new functional relations. There is no regularity in the order or rapidity with which organs and tissues undergo senile involution, and consequently we find vast differences in the mental and physical condition of individuals of the same age. There is a time, generally about the latter part of the seventh or eighth decade, when profound changes occur both mentally and physically. This is the transitional period between old age and senility and corresponds to the critical period that occurs during the period of development called puberty and the critical period during the period of maturity called the menopause in the female. I have called this critical period in the period of decline, the *senile climacteric*. Some at that age show little physical impairment, while others are decrepit. Usually there is mental depression with some impairment of the faculties, lessened activity, and degeneration of some organs and tissues, due to arteriosclerosis or primary degeneration, while other organs and tissues show little change. In those who have lived slow, rational lives, the senile changes proceed slowly, gradually, and harmoniously. Most individuals are so situated or so constituted that greater stress is put upon some organs and tissues than upon others, and these degenerate

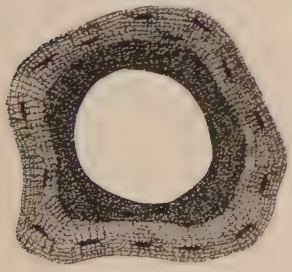


Left femur of a colored man. Large black spaces represent senile absorption of bone.

A single Haversian system, much enlarged, without definite signs of sensility.



A single Haversian system, much enlarged, showing early signs of senility.



A single Haversian system, much enlarged, showing a later stage of senility.

A single Haversian system, much enlarged, showing the latest stage of senility.

Senile changes in the human femur. (J. S. Foote, Smithsonian Miscellaneous Collections, Vol. 61, No. 8.)

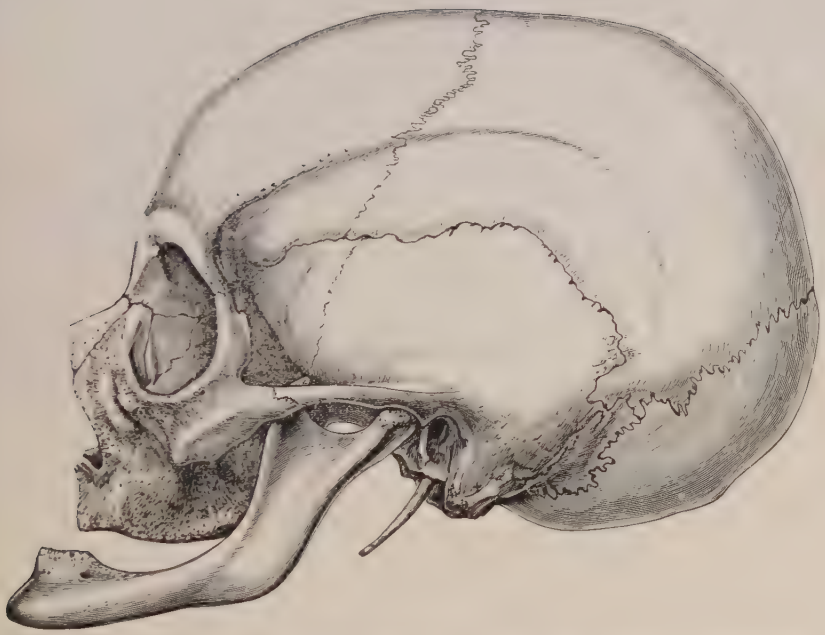
faster than the others. As a result of the unequal rate of degeneration in the organs, the harmonious interaction of functions is disturbed, and we have pathological conditions, giving objective and subjective manifestations of disease. In nature's effort to effect a readjustment of the functions during the senile climacteric those organs which have degenerated slowly now degenerate rapidly, while the degenerative changes in those organs which have been most involved are retarded.

Among the earliest of the obvious changes that occur in the senile climacteric is a change in the mentality of the individual. There is a change in mentality at the beginning of the period of decline, due partly to the recognition by the individual that he is entering upon the closing period of life, and partly to weakening of the intellectual faculties. A more profound change occurs during the senile climacteric. There are now periods of emotional exaltation followed by depression. At times there is mental confusion with delusions which are soon forgotten, flashes of former mental vigor during which brilliant work may be done but if such work is prolonged beyond a few minutes or hours the character of the work deteriorates and it becomes confused and finally it becomes unintelligible, memory is dulled and cannot be stimulated by any process of mnemonics. There are lucid intervals during which there is no evidence of mental deterioration except perhaps weakened memory. Gradually, however, this period merges into the postclimacteric period, the periods of exaltation become less pronounced and less frequent, and the depression gives way to apathy, the reasoning power wanes rapidly, the intense biophilism, or love of life, that marks the early stage of senility, passes away. Interest in all directions is diminished, the individual becomes garrulous, seeks the association of children in preference to adults, and falls into childish ways. Occasionally there is a recrudescence of sexual desire, to gratify which he may attempt rape upon little girls. Such crimes do not arise from depravity, but through weakened mentality involving a weakened moral sense, inability to realize the nature of the act or its consequences, a loss of control over conduct, and an irrepressible sexual fury. Such acts occur almost invariably during the senile climacteric.

Especially noticeable during this period is a change in facial expression, corresponding with the mental change. At the same

time the strength diminishes, and the individual is forced to use a cane; in some cases this is accompanied by senile tremor, rarely by a pseudoosteitis deformans. Owing to the rapid degeneration of those organs which had shown but little senile change before, these organs are peculiarly liable to disease, hence we find most deaths in the early part and middle of the eighth decade resulting from diseases in organs that were apparently healthy before the final illness. While these organs may have degenerated before the climacteric, the process had proceeded so slowly and gradually as to give no subjective or objective symptoms. This is especially noticeable in the heart. If the heart has not been subjected to excessive strain before this time, the cardiac hypertrophy kept pace with the demands made upon the organ. Now, however, it has reached the limit of its ability to compensate for the impaired circulation due to arteriosclerosis and valve defects, and it begins to dilate. In a series of forty-five deaths between the ages of seventy and eighty years, occurring in a fraternal order, there were ten deaths from various forms of heart disease and five from arteriosclerosis.

Other changes that may be noted at this time are the rapid whitening of the hair, where it had thus far retained its color, while the falling out of hair ceases. The skin becomes thin, loose, and transparent; in some cases there is a growth of warts or other excrescences. The dyspnea of senile emphysema frequently disappears as the impaired heart sends less blood to the lungs, thus reestablishing harmonious relations between the two organs. A similar readjustment in the functional relations of allied organs is often found in the activities of the stomach and intestines. The loss of teeth necessitating a change in diet, and change in the functional activity of the digestive organs, possibly, too, a change in the taste for certain kinds of food, cause a change in the nutrition of the aged individual. Insipid articles of food become distasteful. Such substances are usually alkaline in reaction and are indigestible in the stomach owing to the subacidity of the gastric juice. There is generally a dislike for fat and at the same time the secretion of bile is diminished. Underdone meat, a frequent source of constipation, is rejected partly on account of the inability to chew it and partly on account of distaste. Acids and sharp, spiced condiments are relished, and these aid digestion and are of



The skull of a woman eighty-three years old, to show the changes in the mandible and maxilla. (From Morris' "Human Anatomy.")

service in the senile constipation. On account of diminished appetite there are longer intervals between meals, and this prevents overloading the stomach and the addition of food to undigested food already in the stomach. At this time the aged individual demands food in the form of mush or liquid, and softer stools are produced, lessening the danger of fecal impaction and favoring more rapid elimination.

I should ascribe the relief frequently obtained in the postclimacteric period from the trouble of senile constipation of the earlier period to this change in diet and digestion and not to the cathartics that may have been given for years before. The senile climacteric may last a few months or even a year or more. Its inception and completion are gradual, it presents no specific manifestation as occurs in the female in puberty and the menopause, nor are the differences in the organism between the preclimacteric and the postclimacteric periods as marked as between the prenubile and postnubile stages of the period of development or the preclimacteric and postclimacteric stages of maturity. After the senile climacteric has passed, there is a uniform decadence of mind and body. The intellectual faculties become gradually weaker, but rarely reach the stage of complete dementia. Muscle tonicity and nervous activity gradually lessen, breathing becomes slower and more shallow, heart action becomes weak, assimilation becomes more difficult, and elimination is diminished. If no one organ is excessively strained or irritated, the functions maintain their harmonious relations to each other, gradually weakening, until complete cessation in physiological death.

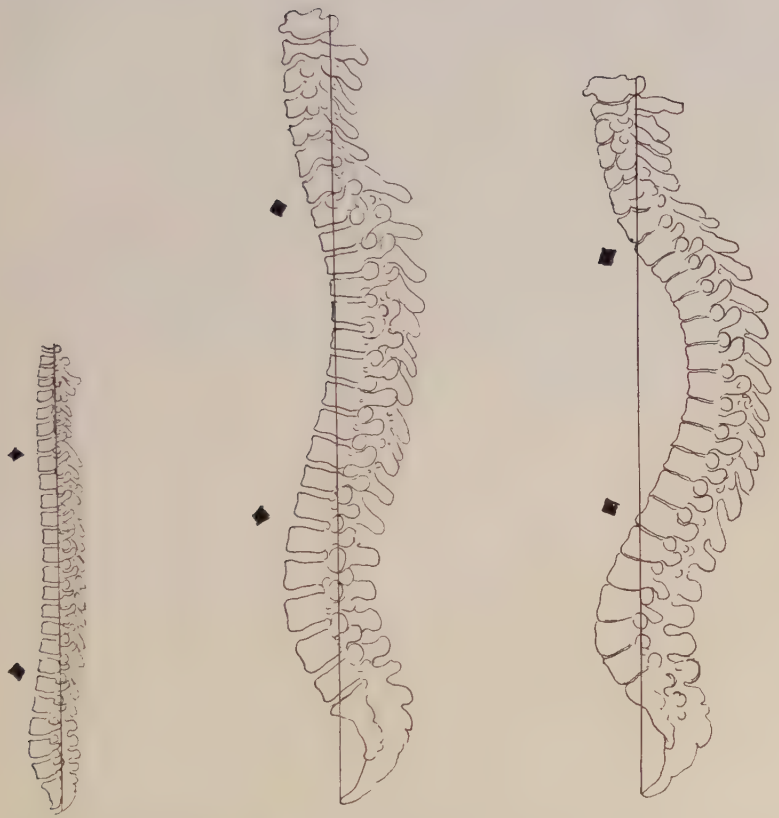
ANATOMICAL CHANGES IN OLD AGE

The anatomical changes due to old age are of the most diverse character, they are neither uniform nor regular nor do we always find like changes in similar tissues in different individuals, until late in life, when changes become uniform and we find like organs subjected to like changes.

The changes in bone include waste of organic matter with consequent proportionate increase in inorganic matter whereby bones become more brittle, they fracture more readily and repair is more difficult; there is osteoporosis of the short bones, of the epiphyses of long bones and of the diploe of flat bones;

late in life there is a waste or resorption of the entire bone substance. Irregular waste and pressure cause changes in the shape of bones. Marked changes are found in the skull, spinal column, thorax, pelvis, femurs and feet.

Cranial bones become thin, local waste occasionally proceeding to the extent of complete perforation, the edges of the opening being raised through the increased osteoporosis of the diploe over the wasted area, and the sutures become obliterated. The most pronounced osseous waste is found in the lower maxilla. Owing to the loss of the teeth and the consequent absorption of the alveolar process, the chin must be raised higher in the act of closing the mouth, the condyles are consequently brought further down and back, the rami become oblique and the angles of the jaw become obtuse. There is at the same time a general atrophy of the bone, the chin becomes more pointed, the mental foramen is smaller and on account of the waste of the body of the bone it is found near the alveolar border. These changes in the lower maxilla produce the weazened face of old age. The changes in the spinal column are due mainly to changes in the intervertebral discs and will be described under cartilage changes, and under the same heading will be found the thoracic changes, The changes in the pelvis are waste, osteoporosis and change in shape, the last being the most noticeable. Owing to the constant downward pressure upon the sacrum this bone is pushed back, the angle of the sacro-lumbar articulation becomes more acute, there is anchylosis of the sacrum and coccyx and the sacro-iliac relations become altered, the ilia being forced backward to accommodate themselves to the changed position of the sacrum. Between this downward and backward pressure of the sacrum and the upward pressure exerted by the femurs the whole pelvis becomes vertically compressed and horizontally expanded. The width of the pelvis is apparently increased still more through waste of the glutei muscles and through changes in the neck of the femur which bring the greater trochanter higher and further out. The ilia become thin, the pubes undergoes osteporosis and late in life wastes, the acetabuli become shallow and larger through the waste of the surrounding bone. The principal change in the femur is found in the relation between neck and shaft. In maturity they form an angle of about 145 degrees, but in old age the neck becomes depressed until the angle



Spinal perpendicular index. Showing relation of length of spinal column to perpendicular line from atlas to tip of coccyx. Infancy, 100 : 95–97. Maturity, 100: 88–95. Old age, 100 : 80–90.

formed approaches a right angle. Osteoporosis destroys the arrangement of the cancellous structure of the neck, the bone being thereby weakened, thus accounting for the frequency of fracture of the neck of the femur in old age. Other changes in the femur are such as occur in all long bones. Broken-down arches of the feet are found generally in the aged. It may be questioned whether this is a physiological or a pathological condition in old age. It is due to the downward pressure upon the feet and weakness of the tendons, and frequently to improper shoes.

The cartilage changes are waste, ossification, calcification and formation of fibrous tissue. The articular cartilages become dry, then thin and through attrition they become fibrillated and waste. In the larynx the thyroid, cricoid and arytenoid cartilages ossify while the epiglottis becomes fibrous. The cartilaginous rings of the trachea sometimes ossify and occasionally the bronchial cartilages suffer likewise. In the sternum complete bony union of the parts of the gladiolus takes place before the thirty-fifth year. About the same time the cartilage between the manubrium and the gladiolus begins to calcify, the ensiform cartilage ossifies and later ossification takes place in the costal cartilages. In old age all these tissues become anchylosed forming with the ribs and spinal column a rigid thorax. The intervertebral discs begin to calcify about the fiftieth year. Owing to the constant downward pressure upon the spinal column when the body is erect these discs become compressed. In maturity the discs are elastic and when the pressure is relieved (as in the recumbent position) the discs resume their natural shape. This accounts for the greater stature in the morning than at night. This expansibility of the discs is lost when they calcify and the diminished stature becomes permanent. The pressure is greatest where the discs are thinnest, anteriorly in the dorsal region and posteriorly in the cervical and lumbar regions. The compression is not so great in the cervical region and the lumbar discs are thicker and more uniform. The greater compression of the anterior portion of the dorsal discs spreads the posterior borders and causes the increased curvature of the spine in that region in old age. It also produces an approximation of the facets whereby a more acute articular angle with the ribs is produced. The ribs in order to accommo-

date themselves to the changed articular relations in the back and the ossified costal cartilages in front become flattened at the sides. These changes, together with the lessened resilience of the ribs owing to the waste of organic matter, occasion the senile chest which resembles the rachitic chest, being longer in front, shorter in the back and flattened at the sides.

The changes in the ligaments are hardening and contraction. The ligamentum nuchæ is sometimes lengthened but never flaccid. The most marked changes are found in the capsular ligaments in which the hardening and contraction may proceed to the extent of complete immobilization of a joint. Stiff joints from this cause are quite frequent but are often diagnosed as rheumatic arthritis. The changes in muscle are atrophy, fatty infiltration, fatty degeneration, stretching and in the case of the heart, hypertrophy.

In active muscles there is primary atrophy through the waste occasioned by muscular action, the waste not being fully repaired. In inactive muscles there is a secondary atrophy following fatty infiltration, the fat cells displacing muscle fiber and appropriating nutrition of the muscles. The difference between the atrophic and the fatty changes is seen in comparing the two biceps of the aged artisan, the right being smaller but maintaining its muscle consistency and strength in proportion to its mass, while the left is flabby, there is but little waste and the loss of strength is greater than in the right. The differences in the changes in the facial muscles between the actor and the dull peasant have been referred to. The greatest waste occurs in the intercostals, these being, after the heart, the most actively employed muscles of the body. The heart muscle hypertrophies, rarely atrophies. Fatty infiltration and fatty degeneration of the heart which are sometimes found in the heart of the aged are pathological and due to impaired nutrition. We sometimes find a pseudohypertrophy of the muscles due to the proliferation of connective-tissue fibers through the muscle fibers. This occurs in the walls of the bladder whereby bands are formed with pouches and pockets between them.

The changes in the skin are of the most varied character including atrophy, localized and more extensive areas of hypertrophy, anemia, congestion, pigmentation and changes in the character of the cells of the various tissues forming the skin.

Section of the Head of the Thigh Bone of a Man of Thirty-seven Years. (From Minot's "Problems of Age, Growth and Death." G. P. Putnam's Sons, New York and London.)

Section of the Head of the Thigh Bone of a Woman of Eighty-two Years. Shows also depression of head of femur. (From Minot's "Problems of Age, Growth and Death." G. P. Putnam's Sons, New York and London.)

There is generally waste of the subcutaneous fat, atrophy of the derma, the areolar tissue becomes fibrillated, the connective tissue becomes loose and separates, the glands waste and there is waste of the elastic fibers. As a result of these changes the skin becomes dry, lusterless, loose and flabby. In some localities the sweat glands exhibit greater activity and the character of the secretion is changed. Pigment is deposited in the rete Malpighii, sometimes localized as ecchymotic spots on the hands, neck and other exposed portions of the body, sometimes covering more extensive areas. In some localities there may be extensive brown patches due to passive congestion. Folds and wrinkles are caused by waste of fat and elastic fibers. The hypertrophies take the form either of thickened epidermis on the hands or feet or there may be hard or soft warts. There is loss of pigment in the hair, the hair bulbs generally atrophy causing falling out of the hair although there is often an abnormal growth in unusual places as in the nostrils, ears, etc. The nails become dry and brittle. The skin is generally cold and where not pigmented it is pale, on account of the deficient circulation, or it presents areas of local passive hyperemia. There is generally degeneration of the nerve terminals producing various sensory disturbances.

The most important senile changes occur in the circulatory system. The hypertrophy of the heart is usually the earliest of these changes but the changes in the blood-vessels produce the most profound disturbances in the organism. The earliest change in the arteries is a hyperplasia of the connective tissue of the intima with consequent stiffening of the vessel, thickening of the inner coat and diminution of the caliber. This is accompanied or followed by a waste of the elastic fibers whereby the elasticity of the artery is diminished. These changes in the vaso vasorum cause diminished circulation through them, interfering with the nutrition of the larger vessels and consequently these vessels degenerate. The inner coat becomes soft, fat deposits and we find atheromatous foci or plaques on the surface of the inner coat. Later results of defective nutrition are: waste of muscle fibers, hardening of the outer coat, calcareous deposits in the inner and middle coats, and finally calcification of the entire vessel. Before calcification the vessel is harder than normal and tortuous. After partial calcification the vessel

feels beady but the pulse can be felt. In diffuse calcification the vessel is rigid and in extreme cases the pulse is absent, the vessel feeling like a hardened tendon. Such extreme cases are, however, rare. Advanced arteriosclerosis is most frequently found in the aorta, the cerebral vessels, the coronaries, the radials, vertebrals, carotids, splenic, brachial, iliac and femoral arteries. The aorta is almost invariably affected, being dilated and showing extensive fatty and calcareous plates in the ascending and often in the transverse portions. The diminution of caliber in the smaller vessels may extend to complete obliteration of the lumen, thereby depriving the parts beyond of nutrition. This causes gangrene or other destruction of tissue. The earliest change in the heart is hypertrophy and this may begin during the period of development. Cardiac hypertrophy which is the normal condition of the heart of the aged cannot be called a senile degeneration, as the same causes that prevail in old age prevail in the earlier periods of life. Whatever tends to make the heart act faster or more powerfully tends to cause it to hypertrophy. Excessive activity, elevation of temperature, nervous influences, will cause hypertrophy, as well as the greater force required to send the blood through the contracted, inexpansible blood-vessels. The heart in the aged is heavier and larger than in maturity, the average weight in the male being 11 ounces and in the female 9 1/2 ounces (Loomis). The cavities are increased, the proportionate capacity remaining unchanged. The left ventricular wall is much thicker than the walls of the other cavities, being thickest just below the level of the mitral valve and diminishing rapidly toward the apex. The valves are thickened and the valvular orifices are enlarged. The aortic opening is larger than the others and aortic insufficiency is the rule in old age. The endocardium undergoes the same changes that are found in the inner coat of the arteries including fatty plaques and calcareous deposits. The more pronounced changes resembling chronic endocarditis will be described under senile endocarditis. Myocarditis, the fatty degenerations, and atrophy of the heart will be treated as diseases. The changes in the veins are similar to the early changes in the arteries. The inner coat becomes soft and there is loss of the elastic and muscle fibers of the other coats. The veins rarely become hard, but the waste of the elastic fibers allows a dilatation of the vessels. They are often

tortuous and occasionally we can feel a venous pulsation, especially in the neck. The veins are usually filled to excess with a slow-flowing current of blood, this condition being known as venosity. The principal pulmonary change is atrophy of the lung. There is diminution in size and weight, the lung is compressed through the changes in the thorax, its expansibility is diminished, the bronchioles show extensive dust deposits partially occluding their lumen, producing pneumokoniosis, the septa between the alveolæ waste and the air vesicles consequently coalesce, producing senile emphysema. The senile lung is grayish with black spots and lines over its surface and throughout its mass, and dilated or ruptured air vesicles are clearly seen on section as minute cavities. The lung has a more elastic feel than in maturity but with diminished crepitation. In advanced senility when the atrophy is very marked they lie close to the vertebral column, their surface is uneven, the upper lobe of the left lung sinks and falls forward of the lower lobe, so that the upper lobe is in front and the lower one is behind it, while in the right lung the middle lobe sinks and falls in front of the lower one. We sometimes find the lower lobe of the right lung overlapping the upper one posteriorly. The respiratory capacity diminishes. The decrease has been demonstrated in the early part of the fourth decade, but not until it is far advanced are marked objective or subjective manifestations produced. The loss, which is about 1/2 per cent. of the total capacity per annum at thirty-five, rises to 1 per cent. or more about sixty. The trachea and the upper part of the bronchi become rigid and frequently contain calcareous incrustations, the bronchioles have their calibers diminished and occasionally the lumen is entirely closed. The pleura becomes thin, dry, lusterless, opaque, the layers are generally adherent to each other and the outer layer is adherent to the chest wall. The changes in the digestive tract are found throughout its entire length. The teeth fall out, the alveolar process of the lower maxilla wastes, the salivary glands atrophy and their secretions are diminished but not altered in composition. The stomach becomes dilated through atony and waste of the muscular fibers, there is an atrophy of the glands, the mucous membrane becomes thin and pale, the amount of hydrochloric acid is diminished and there is probably some change in the character of the peptic secretions. In the intestines there

is a waste of the muscle fibers and diminished secretions. The small intestines become very thin, the large intestines are generally dilated, the dilatation about the sigmoid flexure sometimes forming a pouch or sack, of two or three times its normal diameter. Owing to the impaired circulation hemorrhoids are frequently present. Atheroma of the nutrient vessels causes atrophy of the villi, and waste of muscle fibers causes the folds of the valvulæ conniventes to be smoothed out.

The liver is contracted and harder than in maturity, the cells are smaller, there is an increase of connective tissue and owing to lessened nutrition the organ is paler than in maturity. In extreme old age its weight may sink to 800 grams or less. If, however, there is impeded circulation or weakened heart action there will be engorgement. The surface becomes granular and the capsule becomes cloudy, thick and closely adherent to the surface. The gall-bladder becomes thickened and usually adheres to the adjacent portion of the liver. The duct is thickened and its caliber is diminished. The bile is thicker, more viscid than in maturity and contains a larger proportion of cholestrin. Gall stones are frequently found. The spleen is reduced in size, it becomes firm, the trabeculæ compress the blood-vessels causing them to atrophy and in extreme cases the whole organ is a mass of connective-tissue fibers enmeshing small portions of spleen substance. The relative loss of weight of the spleen is greater than that of any other organ, the spleen in old age weighing less than half as much as in maturity. The pancreas atrophies, there is proliferation with hardening of the connective tissue compressing the vesicles and lobules, the canal of Wirsung becomes hard and its caliber diminished. Occasionally the organ undergoes fatty degeneration without diminution in size. Its texture which is in maturity rather soft becomes harder, of the consistency of softened wax. The kidneys undergo atrophic, sclerotic and other degenerative changes which are so like the pathological changes found in interstitial nephritis that it is often impossible to distinguish between them at post-mortem examinations. The recent discovery of a progressive increase of connective-tissue fibers between the apex of the pyramids—the increase continuing from birth to death in old age—is as far as known the only histological manifestation of ageing which does not appear as a pathological condition.

The kidney in old age has a lobulated appearance, it is granular, pale, and hard, the glomeruli, loops and convoluted tubes atrophy and become sclerosed. The connective tissue forms bands which compress the parenchyma. The capsule becomes thick and adheres closely to the surface. The ureters lose their elasticity through waste of muscular fibers and the tubes become dilated. The inner coat becomes thickened but the caliber is increased and late in life the ureters become stiff fibrous tubes. The senile changes in the bladder begin with a waste of muscular fibers and a proliferation of connective tissue. The waste of muscle permits a dilatation of the walls of the bladder while the connective tissue forms bands in the walls producing constrictions with pockets between them. The sphincter atrophies permitting dribbling. Late in life there is generally a fatty degeneration of the bladder. The prostate becomes enlarged in perhaps one-third of senile cases while atrophy is found in less than 10 per cent. The hypertrophy is in the muscular tissue and there are often found in the mass small fibrous tumors and minute calculi. Sometimes there is an increase of the glandular substance with fatty infiltration. The hypertrophy is generally irregular, often only one lobe being involved. In these cases the favorite location is the middle lobe. When there is atrophy, the changes are similar to the changes in the liver; there is a proliferation of connective tissue which forms bands compressing the gland substance. The ducts are frequently blocked by a deposit of lime salts. The changes occurring in the testicle are similar to the changes in other secreting organs *i.e.*, formation of fibrous tissue bands which compress the glandular substance followed by atrophy and later sclerosis of the substance. The enveloping capsule becomes thick and closely adherent. There is a diminution in the number of the spermatozoa but not in their activity or functional powers. The duct changes are like the changes in other secreting ducts, degeneration and thickening of the inner coat, waste of muscular and elastic fibers, atrophy and sclerosis of the outer coat. The changes in the female generative organs begin at the menopause and while really senile changes they belong to the realm of the gynecologist.

The changes in the brain are atrophic and degenerative. The waste is confined to the cerebrum, mainly at the cortex and most

frequently in the left hemisphere. The loss in weight is about 100 grams at eighty years of age. There is an increase of fluid, a decrease of fat, the brain is denser, there is white softening of the walls of the ventricles, the pia mater is thickened, there are pacchionian granulations and there is an increased amount of fluid in the meshes. The fissures are shallower, the cortex is thinner and frequently contains amyloid bodies while connective-tissue fibers are increased. The nerve fibers are thinner, the cells are atrophied. In many cases minute cavities form around lymph vessels. These cavities called "Etat Criblé," formerly supposed to be perivascular spaces, are retractions of brain substance due to waste. Sometimes spots of miliary aneurysm and softening are found. Atheromatous changes in the vessels of the brain are more pronounced than in any other organ of the body. The changes in the cord are similar to the changes in the brain. Amyloid bodies are frequently found around the central canal, there is a waste of the ganglion cells of the anterior horns, pyramids and posterior fibers. There is a general decrease in volume of nervous tissue, an increase of cerebrospinal fluid, the cord is darker and more firm, the meninges are thickened, cloudy and sometimes contain osseous plates. The atheromatous arteries present a beaded appearance, and there are often spots of miliary aneurysm, hemorrhage and softening. Occasionally zones of sclerosis are found around the blood-vessels, which press upon the nerves causing them to waste. The changes in the nerves are probably similar to the changes in the cord but these changes come on late and the functional changes in the organs usually terminate life before changes in the nerves are far advanced. In the peripheral nerves parenchymatous degeneration takes place in the terminals and terminal fibers and proceed toward the center.

The senile changes in the eye are sclerosis of the lens and weakening of the muscles of accommodation; in the ear there is waste of the drum and a change in the auditory nerves; in the nose there is atrophy of the Schneiderian membrane. The changes in the tactile ends are not well known, but it is evident that some change occurs as there are great functional changes. Neither is it known what changes occur in the taste bulbs.

PHYSIOLOGICAL CHANGES IN OLD AGE

The changes in physiological functions in old age are due either to the anatomical changes or to some causes for which we can find no anatomical change and we therefore call them purely functional manifestations. Many functional changes begin before anatomical changes can be demonstrated and in the nervous system profound alteration of function may persist throughout life, yet postmortem examination does not disclose any change in the tissues. There is no relationship between the extent of anatomical and functional change in old age even in pathological conditions. Neither do the objective or subjective manifestations give any clue to the extent or character of the anatomical or physiological changes. The earliest functional change is diminished activity, diminished power and lessened vital resistance to deleterious influences. Diminished activity is evidenced by diminished metabolism; lessened elimination of CO_2. Diminished power is evidenced by more rapid fatigue or by a greater effort required to do laborious work. This is due to weakening of muscle fibers. Lessened vital resistance is evident from the greater liability to certain diseases, tardy recovery, slow healing of wounds, and the frequency of sequelæ.

Omitting the functional changes due to progressive proliferation of the connective-tissue fibers between the pyramids of the kidneys and those early cases of arteriosclerosis and cardiac hypertrophy which are due to excessive muscular activity, fast living, syphilis and other controllable causes, the earliest functional change occurs in the lungs. Until the limit of growth has been reached the continual deposit of dust in the bronchioles does not impair the respiration. Afterward this deposit diminishes the caliber of the tubes and the amount of tidal air is consequently diminished. Aeration of the blood is now incomplete and from this time forward the imperfectly oxygenated blood has a progressively lessened capacity for carrying nutrition to the organs and tissues and carrying off waste. As this is one of the fundamental causes of ageing, it will be discussed under that heading. The compression of the lungs and their diminished expansibility cause shallow breathing. The vital capacity is diminished at the rate of about 1 1/2 cubic inches per annum and the amount of CO_2 expired

which in maturity is about 1340 cubic inches per hour falls to less than 1000 cubic inches between the ages of sixty and eighty, and may fall to less than 700 cubic inches in extreme old age. The respirations are increased in frequency, there is generally shortness of breath, dyspnea is easily induced, the respiratory motion is confined to the upper part of the chest, the motion being up and down and not expansive. Wheezing and persistent dyspnea are indications of advanced emphysema.

The functional changes in the circulatory system sometimes correspond with the anatomical changes, occasionally there are marked perversions of function which cannot be explained by the changes that are found, and at times physical examination reveals anatomical and functional changes that give no objective or subjective symptoms.

There is high blood pressure as long as the heart can maintain the circulation through a compensatory hypertrophy. When the demands upon the heart are greater than it can respond to, the blood pressure falls below normal. It may also fall through dilatation of the arterioles and when the blood supply to the left ventricle is deficient. The thickening of the aortic valve and the enlargement of the aortic orifice cause an insufficiency of the valve with the consequent regurgitation. This weakens the mitral valve and may cause either rupture of that valve or contraction of the cusps which are already thickened through extension of the senile endocarditis, and mitral insufficiency is produced. The aorta is generally dilated and its elasticity is diminished. These changes cause a delay in the propulsion of blood and the current is slowed, the pulse being normally slower in old age, ranging from 65 to 75 a minute in the male, and from 5 to 10 beats more in the female. Slight influences, however, tend to increase or diminish the pulse rate and permanent bradycardia is not infrequent. The pulse in senility is no indication of the condition of the heart as its strength, frequency and regularity may be influenced by factors outside of that organ. Irregularity in strength and rhythm may exist in the heart without any degeneration of the heart muscle, the fault in such cases being in the nervous regulation. The heart sounds are somewhat altered, the first sound being rough and prolonged and the second sound louder than in maturity. There is occasionally a reduplication of the

first sound due either to irregular contraction of the ventricles or to mitral and tricuspid changes. There is generally an aortic bruit also various valvular murmurs which will be described under the valvular diseases. The blood current is slackened and owing to the contracted vessels the amount of blood in the arteries is diminished. As the changes in the lungs prevent complete aeration of blood the character of the blood is changed and it passes through the capillaries with difficulty. The vis-a-tergo being thus weakened and the vis-a-fronte being reduced through the changes in the right heart and lessened respiratory movements, the veins become filled with a slow-moving current of blood producing venosity and varix.

Investigations into the blood changes in old age give contradictory results and lead to uncertain conclusions. In a series of twelve fairly healthy individuals over seventy years of age reported by Grawitz, the blood count showed red cells from three millions to over five and a half millions; white cells, from 4000 to 8000; hemoglobin from 90 to 110 per cent.; S.G. from 1048 to 1060.

The particular ingredients which are used up in the nutrition of the tissues, and the method of conversion are unknown. It is certain that repair of waste is made up from the serum and that the red cells furnish the oxygen required in the process of metabolism, but neither the microscope nor chemical analysis has revealed how the changes are effected. The simple fact that chemical processes in the body do not correspond with chemical processes outside of the body would indicate that there is a vital factor which modifies the organic processes. Food subjected in a test-tube to the same enzymes that are found in the stomach does not undergo the same change as occurs in the stomach. The character of this vital factor is not known.

With the single exception of the proliferation of connective-tissue cells between the apex of the pyramids, the anatomical changes in the kidneys show no difference under the microscope from the kidneys of interstitial nephritis, but there is a great difference in the performance of the functions. In old age the amount of urine is diminished, it is of lower specific gravity and contains less solids than in maturity, the amount of urea is considerably lessened and may not exceed 125 grains per day, while the amount of uric acid is reduced by about half. There

is occasionally albuminuria which has, however, little significance unless associated with casts. When the urine is alkaline immediately after voiding, it is probably due to retention in a dilated bladder. A dilated bladder will hold urine in the pouches—formed by the contraction of the muscle and connective-tissue fibers in the walls—for days; the urine decomposes and ammonia is produced. The physiological changes in the digestive organs are due partly to the changes in the organs involved, partly to the changed power of assimilation and partly to the food. With the falling out of the teeth solid food cannot be masticated properly nor thoroughly mixed with saliva. Such food is swallowed in pieces, only the outside being acted upon by the saliva and excessive work is put upon the gastric secretions which are already diminished in quantity and probably changed in quality. Solid food is not absorbed unless thoroughly disintegrated and easily soluble. Consequently food, unless introduced into the stomach in such form as to be readily absorbed, remains there for hours, perhaps days, decomposing or undergoing fermentation or it passes into the intestines unchanged giving the intestinal secretions excessive work.

The sense of taste is obtunded, and the muscles of deglutition are weakened so that it requires a sensible effort to swallow. Owing to the anatomical changes in the stomach the gastric digestion is slowed and imperfectly performed. While the dilatation of the stomach and the waste of the muscular coat with the atrophy of the glands lessen the activity of the organ and food remains longer in the stomach than in maturity, weakening and waste of the muscular structure of the pylorus permits food particles to pass into the duodenum unchanged and such particles may pass through the intestines undigested. The rate of digestion in the stomach in old age is much slower than the rate of digestion in maturity.

The most pronounced functional changes in the intestines are constipation, occasional diarrhea, and an excessive accumulation of feces in the colonic pouch. The first of these changes is due to the weakening and waste of muscle fibers whereby peristaltic activity is diminished. This is frequently accompanied by neglect of the aged to attend the call for evacuation of the bowel, and this last is the main cause of the dilatation of the colon and rectum, whereby pouches are formed. Here we see

Ossification of subscapalaris tendon. Waste of bursa. (Courtesy of S. Epstein, M. D., New York.)

one of the many vicious circles which are formed in old age. The diminished elasticity of muscle fibers permits dilatation of the gut which consequently becomes filled with fecal matter distending the bowel, this distention further stretching the fibers and impairing their elasticity. Senile diarrhea, while due to the intestinal changes, is really caused in most cases by improper feeding, the food being taken in too short intervals, in excessive amount, or by entering the intestines unchanged it acts as an irritant.

Functional changes in the brain and nervous system are often the most marked of all the changes that occur in the organism and in many cases there is no corresponding anatomical change. Lessened coordination, slowed afferent and efferent impulses, weakened and often perverted sensibility, impaired activity of the regulating centers and various forms of mental disturbance may be present, yet no morphological change can be found to account for them. These are called senile neuroses. Some of the functional changes in the aged resemble the perverted activities associated with disease in maturity. Senile tremor simulates paralysis agitans but the central canal of the cord may not be encroached upon as is the case in the diseased condition. On the other hand, extensive anatomical changes have been found without functional changes. Bunsen, who died at the age of eighty-nine, was engaged in profound scientific research up to the time of his death, yet his brain was greatly atrophied. The same condition was found in the brain of Mommsen, the great German historian.

Among the earliest of the functional changes in the nervous system are delayed and weakened impulses. Action does not respond as rapidly to the will, and it requires a greater motor impulse to perform the act while greater mental concentration is required to obtain and hold sensory impressions. Brain fag sets in more rapidly than in maturity and while the quality of the work may not deteriorate, the amount of work that can be done at a time is less. Aged writers who could write for ten or twelve hours without intermission during maturity must now take frequent rests else brain fag, then mental confusion and finally complete mental exhaustion set in. The rest must be either in the form of sleep or of some diversion which requires no mental exertion. When an old man falls asleep during a sermon or lecture, it is not through lack of interest but from

brain fatigue following concentrated interest. Tendon reflexes are generally diminished. Ferris and Bosco found the knee reflex absent in 20 per cent., arm reflex absent in 71 per cent., and foot reflex in 81 per cent. of cases between sixty-five and eighty-five years of age. In over 30 per cent., however, the knee reflex was exaggerated due probably to waste of fibers in the pyramids. Sometimes the exaggerated tendon reflex is associated with tremor and a pronounced uncertain gait, the whole simulating cerebrospinal sclerosis. In such cases arteriosclerosis of the brain and cord are usually found and often cerebral softening.

The functions of the sensory organs are impaired in old age. The sclerosis and flattening of the crystalline lens render accommodation for near objects difficult and presbyopia is produced. Where there has been a myopia in earlier life it frequently happens that the senile flattening of the lens will so far reduce the former excessive convexity as to bring about a normal convexity of the emmetropic eye. This explains the so-called "second sight" of aged persons who had been obliged to use glasses in earlier life and can see well without glasses in old age. The term "second sight" is also applied to a myopie condition that occurs in incipient cataract in the aged who have presbyopia or hypermetropia, seeing well at a distance but requiring convex lenses for reading. A swelling of the lens and an increase in its density during the formation of the cataract increases the refraction and the individual can now read without glasses but distance vision is impaired. The acuteness of vision is however not restored, but there is an increasing blurring and dimness depending upon the site, distribution and degree of opacity. Though rather frequent in the aged it is pathological. Weakened accommodation of the muscles interferes with motion of the organ. Owing to the weakening of the muscular fibers of the iris the pupils respond slowly to light, and are generally contracted.

The arcus senilis which is always found in the aged does not interfere with sight nor does it denote fatty degeneration of the heart as was formerly thought. (The author has a well-marked arcus senilis which was shown to the class during his school days.)

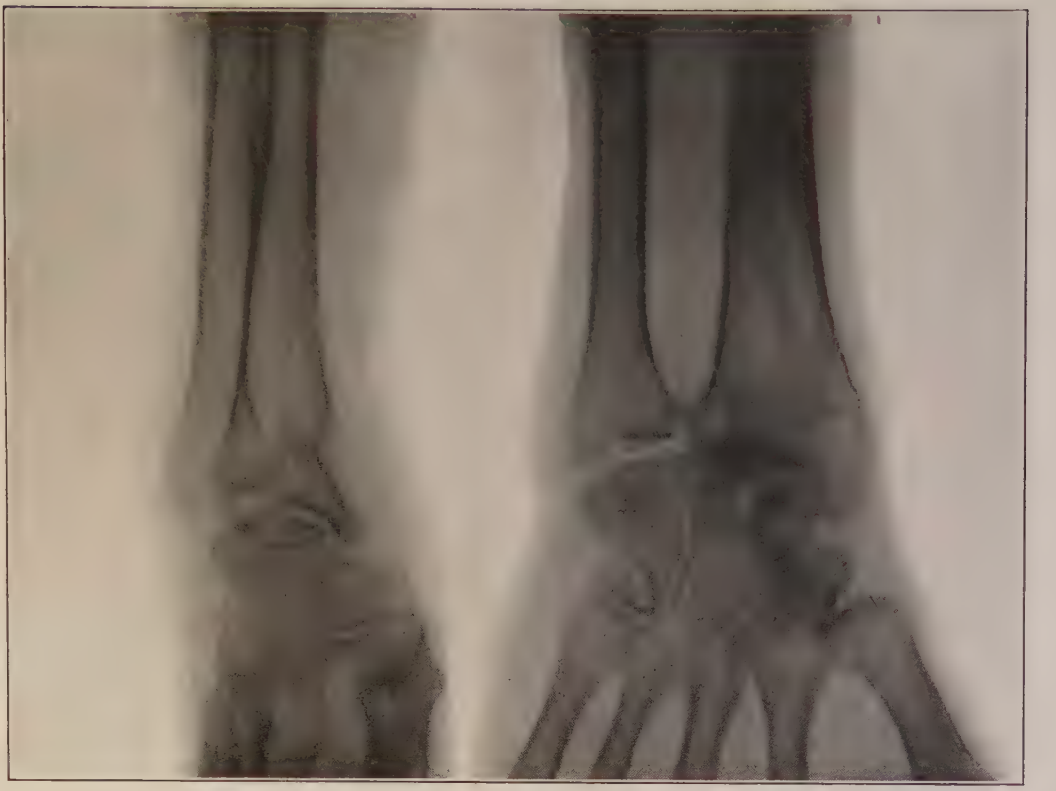
Presbyacusia (deafness) is generally present in old age and in many cases the loss of hearing is complete. This is due to some change in the auditory nerve. The sense of smell is generally weakened and often obliterated. This is due either to the waste

of the Schneiderian membrane or it may be due to atrophy of the olfactory nerve or to a change in the olfactory bulb. There may be perversions of smell for which no explanation can be found.

The sense of taste is obtunded and occasionally perverted. While the sense of smell and the sense of taste probably become weakened through morphological changes in the nerves or end organs, the perversions are probably psychoses. Sensation is impaired in several ways. There may be anesthesia, hyperesthesia and various paresthesias. These changes are due to the changes in the skin and terminals of the nerves. The aged generally feel cold. This is due to lessened surface circulation and impairment of the heat regulation. The weakened tactile sense is due to the degeneration of the tactile end organs. Lessened skin sensibility, anesthesia, is due partly to the mental weakness, the mind failing to note skin sensations unless it is concentrated upon the impression received, and partly to the nerve changes. Hyperesthesia is due to nerve changes. The paresthesias such as numbness, formication, itching, etc., may be psychic, organic, or both.

It is not always easy to say how far mental deviations are natural and normal in old age and where perversion begins. Lessened capacity for work is an early manifestation of senile atrophy of the brain. In some this lessened capacity is shown in rapid fatigue, in some the quality of the work is impaired, in others it is forgetfulness. If one is a writer he finds that new ideas do not come as readily to his mind as formerly; that he must debate over the choice of words, that he must make corrections frequently, while formerly he could write pages without a change. If he is a reader he finds that he does not grasp the substance of his reading readily and that he must frequently read a paragraph several times if he wants to digest its import. He finds it more difficult to concentrate his attention and after reading a few pages he wants to take up some other work. Where formerly a single reading left a clear impression on his mind to be reproduced at will, now the impression soon fades and an effort is required to reproduce it. His interest in general affairs wanes but the interest in a hobby may remain unimpaired or may even be increased as his interest in other directions lessens. He may in this way show greater mental capacity, greater reasoning power than before, but it is all exerted in one direction. The mind is accustomed to activity in many fields.

If all its efforts are directed into one channel it will do more work in this one channel, even though its total capacity for work is diminished. It is simply the principle of economic specialization applied to the mind. This will explain the remarkable works turned out by great men in all fields in their old age. Where there is general mental decay, the will, sensations, intellect and emotions become less active, the weakening being progressive until complete dementia is reached, and the individual's existence is like that of the absolute idiot. In these extreme cases, however, there is usually pathological cerebral softening. The change in temperament which is often marked in old age is not a senile psychosis though generally classed among the senile psychic changes. It seems to me that it is the natural result of the realization of advancing age with diminished powers, lessened opportunities, increasing discomforts, and the fast approaching termination of life. Similar temperamental changes are observed in the young when they find that they are suffering from a fatal disease. The moment a man becomes a grandfather, though he be but forty, he begins to feel old and changes in his temperament and demeanor can be noted. Other causes such as a sudden fright, a secret fear, a great loss, will do the same. Owing to the weakened intellect in old age the individual loses control over the emotions, weakened memory, especially for recent events, makes him more conscious of the old order of things, he becomes "old fashioned," holding on to ancient ideas and methods, and becomes irritated when these are displaced. These idiosyncrasies become obnoxious to the younger generation and they look upon him as queer. The idiosyncrasies become more pronounced when the old man grows careless about his person and his surroundings, although this is mainly due to his desire to avoid everything that may cause physical exertion. Even among old women who were formerly extremely neat this carelessness about their surroundings is often noticed. Owing to their innate vanity they may, however, present an appearance of neatness though often this applies only to externals. Among the depressing influences of early senility is diminution of the sexual powers without diminished desire. Where desire and power diminish together this is not noticed, but the loss of the power alone often leads to the sexual perversions of the exhibitionists. Mental weakness produces an expression of apathy; in mental depression there is



Osteoporosis and waste of cancellous structure of lower ends of radius and ulna. Hardening of radiocarpal ligaments simulating fibrous adhesions. (Courtesy of S. Epstein, M. D., New York.)

moroseness, irritability, often pre-occupation (day dreaming), the individual talks to himself, sometimes exhibiting anger or fear upon the slightest provocation or without any apparent reason. (The changes during the senile climacteric have been described in the chapter on The Senile State.)

CAUSES OF AGEING

The question why we grow old, or rather why, after a period of physical perfection the organs and tissues degenerate and their functions become weakened and perverted until they are unable to maintain the harmonious interrelations necessary for life, is part of the great problem of life and death. Natural phenomena require scientific explanations. Recourse to unnatural, supernatural and superhuman agencies, a belief which can only be based on faith, is simply an evasion of a scientific explanation. Of the metaphysical trinity—the beginning, life and the hereafter, life is the one which is tangible to the extent that its manifestations may be studied. The nature of the force which we call vitality is unknown, but we cannot say that it is unknowable. A scientific theory of life must have a comprehensible basis though we may not be able to prove the theory or the existence of the basis, with our present methods of investigation. When we fall back upon "the will of God" or "fate" or "nature" as our explanation of life and its manifestations, we accept the basis on faith and not on fact or reason. We know that life depends upon the existence in the protoplasm of a property called irritability. When that property is absent the protoplasm is only a highly complex chemical which we have not yet succeeded in making synthetically. Ageing is a manifestation of life. It is found throughout the animal and vegetable kingdom; trees show the effects of age and even among the infusoria there are changes which result in their death and which are considered to be senile degenerations. Many theories have been advanced to account for this ageing. The old idea that the body becomes worn out like an old engine and the tissue wastes just as the material wears away in machinery or goods, is a fanciful simile without a basis of fact. There is no similarity between the wear and tear of material through friction or by the elements and the waste of tissue, except in the wear of the epidermis. Neither does the body become worn out through activity, indeed in the early period of life activity hastens repair and growth.

There is no analogy between the metabolic processes and the wear and repair of inanimate things. Organs and tissues that are rarely or never used in one individual undergo the same degenerative changes that occur where they have been actively employed in another. The virgin uterus undergoes the same change at the menopause that occurs in the uterus of the multipara. The brain of the absolute idiot shows in advanced age the same changes that go on in the brain of the sage. In old age repair is not as active as in youth and excessive activity hastens waste, but this is the result and not the cause of the senile changes. There are two prominent histo-mechanical theories; that of Demange and the more recent theory of Thoma. Demange ascribed the cause of the senile changes "to a change in the quantity and quality of the interstitial nutritive material due to changes in the circulation and this is due to atheroma and arteriosclerosis." Tracing back the cause of the vascular changes he believed that the constant friction of the blood against the inner coat of the vasa vasorum irritated the endothelium, this persistent irritation producing an endarteritis, with thickening of the inner coat and consequent diminished caliber of the fine vessels. The supply of nutrition to the larger vessels being thus diminished they begin to degenerate through fatty infiltration and production of atheroma. The subsequent senile changes are due to diminished nutrition following diminished caliber of the nutrient vessels. This theory followed the work of H. Martin on the pathogenesis of atheroma. Martin found cases of obliterating endarteritis of the vasa vasorum followed by a necrobiotic infarct in the larger vessel. The affected cells excite further irritation in the surrounding tissue of the vessels, producing various forms of degeneration, thereby interfering with the circulation through them. Thoma believes "that the ceaseless activity of the heart and blood-vessels gradually weakens the elastic fibers of the vessels as would happen in a piece of rubber which is expanded and contracted with ceaseless regularity. The loss of tone thus occasioned permits a dilatation of the vessels, the circulation is slackened partly through the dilatation and partly through the lessened contractility of the vessels and the nutrition of the organism is thus impaired." There are several other theories giving nutritional changes following vascular changes (atheroma and arteriosclerosis) as the cause of ageing,

differing, however, from Demange as to the cause of the arterial degeneration. The principal objection to the theories which charge the senile changes to arteriosclerosis is that arteriosclerosis occurring in maturity in connection with other diseases and even without any other pathological condition is not followed by such changes as occur in old age. Another class of theories is based upon the assumption of the existence of a vital principle. This assumption has no demonstrable basis. If there is a concrete something, which is the essence of life and which causes all the changes that constitute life, we can form no conception of its character. If we say it is the soul we have again an inexplicable, intangible, immaterial something not susceptible of scientific proof. Vitality itself may be a form of energy not bound by the physical laws with which we are familiar, since no other form of energy can be converted into it, and it is subject to final extinction. This, however, does not justify the assumption of a vital principle, nor is there any other scientific basis for such assumption. Durand-Fardel's theory was based upon the existence of a vital principle of limited duration. He believed that in animal life, as in some forms of plant life, when the organism had completed its purpose to reproduce the species and thus perpetuate the race, there was no further object in its existence.

After the menopause in woman and the critical period of man, heteroplastic processes take the place of the normal anabolic processes and these lead to such changes in the functions that their harmonious interaction is impossible and death results. It is hardly necessary to show the fallacy of this theory since physical life is not dependent upon sexual activity. The most prominent theory of ageing to-day is Metchnikoff's theory or rather theories, of tissue phagocytosis and autointoxication through the absorption of the products of intestinal decomposition. The theory that the waste of tissue in advanced age is due to the destruction of the tissue cells by macrophages has been criticized on the ground that in many tissues where there is extensive waste no macrophages are found and the waste can be explained by insufficient nutrition. Even in the brain where Metchnikoff demonstrated the presence of neurophages in the aged, atrophy has been found without neurophages. The theory of autointoxication through the absorption of ptomaines and other toxic material from the intestines, which the organism

is unable to eliminate, is even less tenable. While such absorption undoubtedly takes place in old age it also occurs in maturity and youth, without producing senile changes. Defective elimination of leucomaines and other waste products of metabolism has been given as a cause of ageing but the criticism applied to the autointoxication theory of Metchnikoff, applies to this. A discussion of all the theories and the many theoretical and practical objections to them would carry us beyond the purpose of this work. Some theories are based upon some change in the character or composition of the blood, yet there is no uniformity in the changes of the blood in old age. A recent theory of this nature is based upon unstable metabolism. This causes a change in the blood salts and a consequent abnormal diosmosis, which impairs the circulation. The changes in the blood irritate the lining of the blood-vessels causing arteriosclerosis, which further interferes with the circulation and the nutrition of the organs and tissues.

Sir Victor Horsley's theory that ageing is due to degeneration of the thyroid gland, and Lorand's elaboration of that theory including other ductless glands, do not give us the cause for the senile degeneration of these glands and ageing may occur without demonstrable changes in these glands.

Naunyn presents a double theory, waste and wear of tissue during activity, which is not fully repaired, and weakening of the heat-regulating centers. He points out the deficiency in the organism to repair the loss of elasticity in the arteries caused by activity, and sees therein a general fault in the organism to make good the losses occasioned by activity. The virgin uterus at the menopause and the brain of the idiot in old age dispose of that part of his theory. He also says that the local loss of heat from the surface causes general loss of heat. In maturity this loss is replaced through muscular activity, and this in turn creates the necessity for food to replace the waste of muscle. In senility the heat regulation is weakened and the loss of local heat does not arouse the same necessity for muscular activity nor is there the same desire for food. Lessened food produces lessened nutrition to repair waste and less fuel for combustion. The objection to this theory is that lessened muscular activity is due to the anatomical changes in muscles and joints making motion more difficult, and that the desire for food, though stimulated by muscular activity, exists without the necessity for such stimu-

Colonic Pouch. (From Tyson and Fussell's "Practice of Medicine.") Photograph of colonic dilatation in a young man. Used here to illustrate the senile colonic pouch, which it resembles.

lation as is seen in the paralyzed person, while forced feeding in old age does not increase the repair of waste. The slightly lower temperature found in the aged can be accounted for by the lessened muscular activity and also by lessened metabolism. (The senile individual requires only from 75 to 80 per cent. of the calories required in maturity.) The diminished surface temperature is due to impaired surface circulation and changes and not to weakened heat regulation. There are several theories based upon cell changes. Ever since Schwann presented his famous cell theory, investigators have sought in the cell the solution of the problems of life, of birth, of growth, decay and death. The earliest of the cell theories was presented by Canstatt. He believed that the cause of ageing was to be found in the cell, that the death of the cell was so much molecular death of the organism which was not replaced. Canstatt did not take into consideration the repair of tissue through cell reproduction or that in the course of a long life probably every cell that existed at birth had been destroyed and replaced repeatedly. Minot gives as his theory of ageing an increase in the protoplasm and a differentiation of the cells.

The theory of tissue-cell evolution which I advanced as the fundamental cause of ageing is based upon some facts and some assumptions. I believe that there is a progressive evolution in cell life; that newer cells differ from their predecessors; that at one stage of this evolution the cells are most perfectly adapted to their surroundings and their available nutrition; that at this time the cells are in the most perfect condition to perform their functions; that later cells are less perfectly adapted to the conditions under which they exist; that under these circumstances fewer and more imperfect cells are formed; that finally the cells are so imperfect and so poorly fitted for the conditions under which they exist that they either do not reproduce or else the organs which they form cannot perform their functions. Furthermore, different kinds or classes of cells have different stages or periods of evolution, but tissue cells of the same kind pass through the same stages.

We know little of the properties of cells. They have the power of selection of nutrition, of assimilation, of excretion, of growth and reproduction, and some have the power of motion. The why of their activity is as much a mystery as is the source of their vitality. May it not be that in the course of the con-

stant destruction and reproduction of cell life that is going on in the organism there is also going on a constant evolution in cells? It would be analogous to what is going on in the more complex forms of life. No animal reproduces its exact counterpart. There is a difference in oysters coming from the same spawn, in trees growing from the seeds of the same apple. Under the microscope a whole colony of germs of the same variety may look alike yet under the influence of a germicide some will survive longer than others. If they possess greater vitality it is because they possess qualities or properties making them more resistant to deleterious influences.

There is a difference in the properties of the protoplasm of different kinds, and to the difference in the proteid molecules is due their diverse activities. They differ in their special properties, the performance of specific functions, and the general cell properties, growth, assimilation, etc. They also differ in their tenacity of life, in their resistance to unfavorable influences, violence, temperature changes, change of nutrition, phagocytes, etc. Slight differences may be found in tissue cells of the same variety taken at the same time from the same tissue, but we will never find the great differences that exist at different evolutionary stages of the same tissue; that is to say, we will never find at the same time, for example, degenerate senile cells of the ovary and the early immature cells, though we may find, during the menopause, active cells and cells showing early degenerative changes. Of course, under abnormal disease conditions cells of different stages of evolution, imperfect cells, even cells of entirely different character may be found together. The difference in the length of the period of evolution in different cells can be shown by comparing the cells of the thymus gland with connective-tissue cells. The first indication of this gland is a faint line which appears about the tenth week of intrauterine life. It grows like all tissues, through cell growth and reproduction, the cells becoming larger and more numerous. The cells formed about the second or third year of the person's life are best adapted to the available nutrition, to their surroundings, and to the conditions under which they exist. The cells and the organ have now reached their limit of size and activity. No satisfactory explanation has yet been given for limitation in size, whether it be cells or elephants. To account for the retrograde evolution of the thymus we must reason from analogy.

At the time that the cells are in their most perfect condition they receive an ample supply of nutrition from the blood and as there has been a constant increase in the rate of reproduction newer cells are formed more rapidly than the waste of old cells. But the limit of available nutrition has been reached. The amount of blood has increased, but with the change in diet and the increase in physical activity there has taken place a change in the condition and composition of the blood, it does not contain enough of the particular ingredients necessary to supply the thymus cells. The newer cells are consequently not in such perfect condition as their predecessors, they are more readily destroyed and they reproduce more imperfect cells. The cells are now in the stage of retrogression, fewer and more imperfect cells are formed until about the period of puberty or a few years later when the cells become extinct. The entire period of evolution of the thymus gland cells is from about fourteen to twenty years.

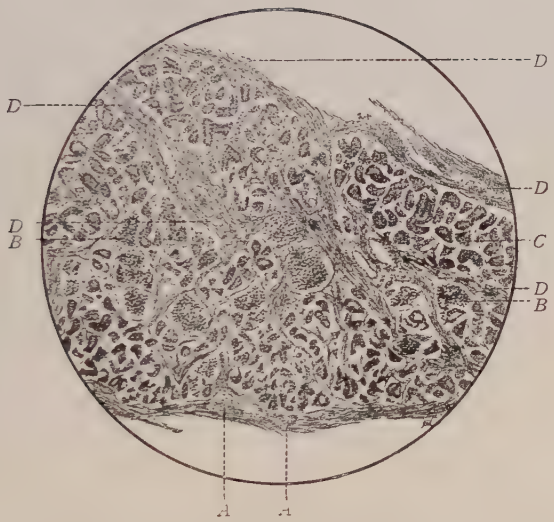
The evolutionary changes in the connective-tissue cells proceed so slowly that it is not until late in life that any marked change in them is observable and then it is an increase in rate of growth and reproduction. It is impossible to follow up the evolutionary stages of connective cells as the destruction of their microcosm, the individual, ends their career while in the stage of active progressive evolution. Let us apply this theory to the ovarian cells. From the beginning in intrauterine life there is a slow, steady growth in number and size of the tissue cells, until at the time of puberty they are most perfectly adapted to their surroundings, their nutrition, and the purposes of the immature organ, but they are not adapted to the great purpose of the ovaries as reproductive organs. The period of puberty is a transitional stage in the evolution of ovarian tissue cells, the evolutionary changes proceeding rapidly, the newer cells having functional properties not possessed by their predecessors, the older inactive cells disappear, and by the end of that period only new, active cells remain. For the next thirty years, the period of sexual activity, evolutionary changes proceed slowly, for years they are apparently stationary. Toward the end of the active period the organ becomes less active but is still able to perform its functions. The menopause is another transitional stage. The old cells are still active but the newer cells have not the same properties as their predecessors, the activity

of the organ decreases with the gradual destruction of the old active cells, and at the close of this period the organ is composed of cells having different properties from the cells of the active period. These new cells are not perfectly adapted to the conditions under which they exist and they reproduce cells still less fitted for those conditions. Fewer and more imperfect cells are formed, and the organ shrinks. The late cells are very tenacious of life and persist throughout the life of the individual.

While cells of the same variety pass through the same evolutionary stages, there may be wide variations in the length of time required to pass through each stage. Hyperplasia of connective tissue may begin in the thymus gland in the second or third year, when that organ first begins to retrograde, in the ovary at the menopause, in the brain at advanced old age. Puberty changes may occur in one person at eleven or earlier, in another at sixteen.

This theory is opposed to the theory of the sudden conversion of one variety of cell into another variety of cell, as the passive cell of the hair cylinder into an active phagocyte; it is not, however, opposed to the rapid conversion through intermediate cells, as the ovarian cells of the anteclimacteric period into the cells of the postclimacteric period. Pathogenic causes may produce sudden and revolutionary cell changes, but such are not considered in normal processes. This theory of tissue-cell evolution will explain the greater resistance to disease and deleterious influences at one period of life than at another and in one tissue or organ than in another, the formation of pathological processes and their preference of location, morbid selection, perverted activity, and the whole cycle of the processes of life from its inception to its dissolution.

The most important factor in the life of the cell is its nutrition, which is derived from the constituents of the blood. Impairment of the blood, either through the presence of abnormal constituents or some alteration in quantity or quality of the normal constituents, affects only those cells which derive nutriment from those impaired constituents or assimilate the toxic material. These cells are weakened, their activities are impaired, and they reproduce defective cells. With the improvement in nutrition and other conditions necessary for their existence the cells improve and reproduce more perfect cells. This is the process of recuperation. In senility the cells of most



Pancreas Showing Increase of Fibrous Tissue. Chronic Interstitial Pancreatitis. (From Coplin's "Manual of Pathology.") *A, A.* Areas of hemorrhage. *B, B* Immature gland cells (bodies of Langerhans). *C.* Gland acinus. *D, D, D, D, D.* Fibrous tissue; the areas of rhexis (*A, A*) are also in the fibrous tissue.

tissues are retrograding, and they are not well adapted to the available nutrition and surroundings. If there is any perversion of this nutrition, cell destruction proceeds faster, reproduction is either abolished or the new cells are so defective that the organs which they form can barely perform their functions. With an improvement in the nutrition there may be an improvement in the cells, but the blood in old age does not furnish the best nutritive material to senile cells and these cells reproduce cells still more imperfect than themselves. The altered metabolism in old age is due to the altered properties of senile cells. Late cartilage cells seem to have a "liking" for calcium salts, that is to say, having the power of selection of nutrition they absorb such salts from the blood more readily than the cells at an earlier period. Other senile cells show the same tendency. Late cells seem to be tenacious of life for they effectually resist bacteria and other agents destructive to earlier cells.

According to this theory the vital resistance of the individual, that is the power of the organism to resist certain pathogenic influences, is increased in old age. This is diametrically opposed to the generally accepted view that the vital resistance in the aged is diminished in all directions, yet it is well known that infectious diseases are rare in old age, that acute inflammations rarely occur and that most diseases that are apt to occur in the aged, except those arising from perversions of the normal senile processes, are modified and less active at that period of life. It is evident then that either the aged cells offer greater resistance to pathogenic germs or that phagocytosis is more active. The latter would account for the milder attacks of infectious diseases the former for their rarity. Probably both factors prevail in old age. Disease in old age is generally due to some perversion of the normal degenerations due to age, or to a disturbance in the harmonious relations between the functions, caused by a more rapid degeneration in one organ than in a co-related organ. Where the harmonious relations are maintained to the end, physiological death ensues without disease. In such cases death is due to diminution in nervous activity to the point of complete cessation.

Senescence is not due to any one cause. There is undoubtedly a determining factor which is the subject of the various theories that have been advanced, but there are in addition

contributing factors, causative and resultant, which hasten the senile processes. With the first breath that the infant draws it inhales dust and the foundation of pneumokoniosis is laid. During the period of lung growth the increase in substance and size of the vesicles prevents any impairment from the deposit of dust in the tubes, finer bronchioles, alveoli, lymph spaces and connective tissue. After the lungs have ceased to grow the deposit continues, the lumen of the finer tubes is diminished, the dust sets up a pulmonary fibrosis, less air passes through the lungs and the blood is insufficiently oxygenated. Imperfectly aerated blood causes imperfect nutrition and imperfect elimination of waste. In addition to this there are going on in the chest walls senile changes which compress the lungs, diminish their expansibility and thereby further impair the respiratory functions. The greater effort required to get sufficient air into the lungs, shown by the increased number of respirations, puts a greater strain upon the respiratory muscles, and the greater activity aroused increases the waste which is not fully repaired owing to the vitiated condition of the blood. At the same time the greater effort required to force sufficient air into the lungs and the impaired nutrition of the vesicular walls weaken the walls of the latter, they become thin and finally rupture producing senile emphysema with diminished aerating surface, increased residual air and lessened vital capacity. In the meanwhile changes have been going on in the heart and blood-vessels, the blood supply is diminished and later when the heart becomes weakened the pulmonary circulation is weakened, still further impairing the oxygenating function of the lung while the impairment of the bronchial artery and the vitiated blood prevent the complete repair of the lung tissue, which wastes more rapidly in its efforts to carry on its functions under difficulties. In this resume of the changes that go in the lungs in ageing we find as the most important contributing factor a pneumokoniosis that began at birth. A dust-free atmosphere is theoretically conceivable but practically impossible. It is true that most of the dust inhaled is caught in the cilia of the bronchi and is expectorated but some does reach the finer vessels, gets into the lymph channels and into the substance of the lung. This is inevitable. This pneumokoniosis is not to be considered in the same light as the vocational dust diseases,

as the latter are entirely independent of the senile changes and cause a controllable irritation in the lung which leads to lung destruction.

Other contributing factors to the senile changes cannot be even theoretically remedied. In the spleen, for example, there is proliferation of connective-tissue fibers, some of which compress portions of spleen substance causing it to atrophy, and some of the fibers crossing blood-vessels compress and finally obliterate them thereby depriving the spleen substance of nutrition.

Disease is not a causative nor even an essential factor in physiological death, for disease is unnatural and in the nature of an accident. Physiological death occurs without a perversion of function or structure which is the essential element in disease. Sanitation, hygiene and dietetics serve to prevent disease but they have no influence in prolonging life aside from the prevention of disease. The longevity of the Jews under the most unsanitary conditions is proof of this statement. Other long-lived nations such as the Bulgars, Roumanians and Russians reach an advanced age in utter disregard of sanitary regulations.

The prevalence of precocious senility in nations which subject themselves to intense mental and physical activity strengthens the theory of tissue-cell evolution as the determining cause of ageing. This excessive activity hastens the evolution of the cells, by causing greater activity with consequent more rapid destruction and reproduction and the earlier appearance of cells of the later evolutionary periods. I have not touched upon the factor of heredity as I do not believe that heredity has any direct influence upon longevity. Healthy parents beget healthy children and if the children live under the same conditions as their parents, they have the same chance to reach old age. The farmer's son who remains on the farm will age more slowly than his brother who goes to the city.

The theory[1] of tissue-cell evolution is in accord with what we know of evolution in the higher and more complex forms of life. What the eon is to the universe and the geological period is to mundane life the years of an evolutionary stage are to the cell. A gradual and progressive process, cell evolution is the natural conclusion that has cosmic evolution as its major and race evolution as its minor premises.

[1] This theory of senescence was first published in the New York Medical Journal, Nov. 5, 1910.

W. B. MEACHAM.

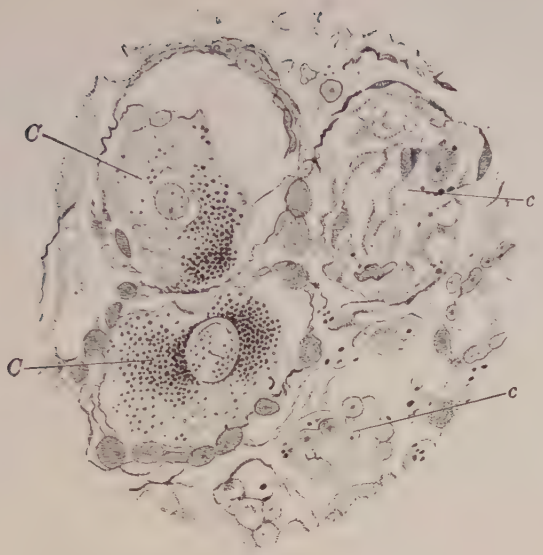
PART II

PATHOLOGICAL OLD AGE

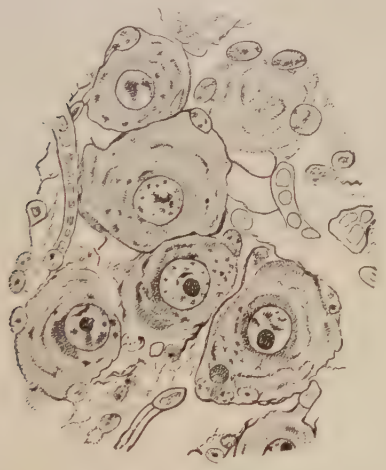
GENERAL CONSIDERATIONS

Disease in old age must be looked upon not as a pathological process in an organ or tissue such as we find in maturity complicated by senile degenerations, but as a pathological process in a normally degenerating body, and the perversion of function is not a perversion from the normal functions of maturity but a perversion from the functions that are normal to the degenerating body. This conception of disease in old age will lead to a discriminative valuation of the symptoms and signs of disease and the manifestations of senility. The latter are often more pronounced than the symptoms of disease and unless we can separate the manifestations of normal senile processes from the symptoms of disease and eliminate the former from our diagnosis, we will treat an incurable, progressive, physiological degeneration while the disease is killing the patient. Not infrequently the manifestations of old age simulate the symptoms of a disease and the patient is treated for a pathological condition which is not present. Another frequent source of error in diagnosis is due to obscure symptoms which may be unnoticed or uninterpretable. This is often the case in senile pneumonia, the symptoms of which are at times so mild as to arouse no suspicion of the grave pathological condition present until pulmonary edema sets in. Another source of error in senile cases is in the interpretation of symptom complexes. We often find in maturity a number of symptoms which taken collectively are diagnostic of a certain disease. In senility every symptom must be traced to its source before we can determine its relation to the disease which we suspect. In senility the severity of symptoms bears no relation to the severity of the disease nor does moderation of symptoms always denote an improvement in the pathological conditions. A fall

in temperature may be due to exhaustion which ends in death. A rapid fall in blood pressure may be due to sudden weakening of the heart, the sleep after delirium may be a coma from which the patient will never be roused. A rapid loss of weight may be due to waste of fat and the wrinkled skin after such loss gives the individual the appearance of age. A rise in temperature is invariably due to disease and the more rapid the rise the more serious the disease. If a chill precedes the rise in temperature we have a grave condition which will probably end fatally. Temperature changes are, however, uncertain indications of the character or severity of a disease, and unless the temperature is taken in the rectum it is misleading. Even in health in the aged there may be a difference of two degrees between the rectal and axillary temperature and the same difference can be found in the mouth at different times. It is not unusual to find a mouth temperature of 97 or less in perfect health. Elevation of temperature as the result of disease proceeds less energetically, more slowly, and does not rise as high as in the same disease in maturity. If the mouth temperature is taken as is generally done in adults, we are liable to be several degrees out of the way and we will get an intermittent or septic temperature range due to local causes not connected with the disease. The typical temperature curves associated with certain infectious diseases are rarely seen in old age. It cannot be insisted upon too strongly that temperature in the aged should be always taken in the rectum only. The pulse in old age is worse than useless as a diagnostic agent, for it will often deceive us. As arteriosclerosis is almost always present, the artery is harder than in maturity and we get a hard pulse that may simulate the pulse of serous inflammations. We may get this pulse in aortic stenosis in which condition the pulse is usually small, slow and weak and we may get the same pulse in aortic insufficiency in which disease in maturity we get the water hammer pulse. There are many conditions beyond the aortic orifice to modify the radial pulse and destroy its value as a diagnostic agent. Blood pressure in the aged has not been studied sufficiently nor have similar findings received similar interpretations. As we have in almost every senile case arteriosclerosis and contracted kidney, two causes for increased pressure, high readings are physiological but no standard for



Group of Four Nerve Cells from the First Cervical Ganglion of a Man Dying of Old Age at Ninety-two Years. Specimen preserved with osmic acid. *C*, *C*, two cells still intact, but loaded with pigment granules; *c*, *c*, two cells which have disintegrated. ×500 diams. (From Minot's "Problems of Age, Growth and Death." G. P. Putnam's Sons, New York and London.)



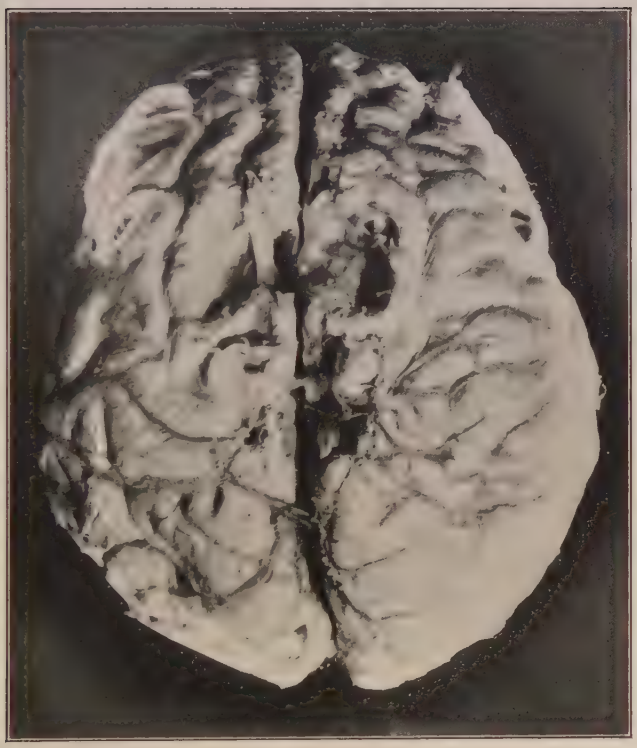
Group of Five Nerve Cells from the First Cervical Ganglion of a Child at Birth. Specimen preserved with osmic acid. ×500 diams. (From Minot's "Problems of Age, Growth, and Death." G. P. Putnam's Sons, New York and London.)

senile cases has been established nor are investigators agreed upon the meaning of deviations from the usual findings in the aged. The closest approximation of a standard of normal blood pressure in the aged is the age plus 100 in m.m. The face gives us little information in senile cases. There is the expressionless face of senile dementia and of paralysis agitans, the latter disease being readily distinguished from the former by the tremor and brighter mentality. There may be jaundice indicating biliary obstruction or cancer; in apoplexy the face is puffed and congested; in chronic nephritis puffy and muddy or pasty looking; it is flushed in hyperemia and fevers, pale in anemia, sallow in various cachexias; but these facial indications have only a secondary value. Almost every one who has reached advanced age has led an out-door life, his skin is tawny and weather-beaten and does not readily show these changes. The contracted pupils which are normal to old age may mislead us, the dribbling of saliva may suggest salivation, occasionally one sees the stare which, associated with contracted pupils, is found in mania. This will be found in the aged individual who has presbyopia and does not wear his glasses when he makes an effort to see a speaker close by. Ordinarily the aged patient is apathetic or if he realizes the seriousness of his condition he is anxious or depressed. In most diseases having a fatal outcome, the mind becomes dull and as the end approaches the patient becomes unconscious. Pain is an uncertain symptom in old age, as it is frequently referred to some organ or tissue not diseased and it is often absent or slight in diseases in which pain is usually a prominent symptom. The absence of this symptom in pneumonia, gastritis, peritonitis, etc., may lead to a wrong diagnosis or to the neglect of the disease by the patient himself until death ensues. Gangrene seldom gives pain and it may be neglected until extensive necrosis has occurred. This absence of pain in senile diseases is usually associated with weakened mentality and it is probable that the mental condition is responsible for the lack of appreciation of painful sensations, as well as the condition of the nerve terminals. There may be on the other hand hyperesthesia and paresthesia, especially itching, so severe as to require medical attention, yet no pathological lesion can be found. The altered reflexes in old age frequently make a correct diagnosis

difficult and if the disease is one in which the state of the reflexes is diagnostic or confirmatory of a diagnosis, it is almost impossible to avoid error. Investigators have found the foot reflex absent in over 80 per cent. of cases between sixty-five and ninety-three years of age, yet the knee reflex was increased in 32 per cent. and absent in but 20 per cent. These findings show the unreliability of the state of the tendon reflexes as a diagnostic aid in old age. If there are two conditions present which ordinarily give different reflexes we must omit the tendon reflex entirely in determining our diagnosis. A frequent source of error in diagnosis in senile cases is the changed position into which organs are forced through anatomical changes in other structures. The flabby abdominal muscles and weakened diaphragm permit the liver to sink until in exceptional cases the upper border can be felt below the ribs. In these cases the organ appears to be much larger than when the outlines can be determined only by percussion. The stomach also sinks when the abdominal walls are flaccid and the intestines are empty, but when the bowels are filled with flatus the stomach may be raised or pushed to one side. Owing to the rigid chest walls and wasted intercostals the apex beat may be quite pronounced even in the case where the heart is weak as in cardiac dilatation. The weakened diaphragm allows the heart to sink until the apex is 3 inches below the nipple and further to the left than in maturity, yet a dilated stomach or intestines distended with gas can raise the diaphragm and push the heart further up and to the left. In determining the meaning of an abnormal position of the heart, the condition of the stomach and intestines must be taken into account. Owing to the rigidity of the chest walls inspection gives us little information of pathological conditions within the walls, while the up and down respiratory motion is apt to mislead us. In senile pneumonia the apex is generally affected, but the lungs in old age are atrophied and we must look for the altered percussion note in the infraclavicular space. Slight bulgings between the ribs are generally due to pleuritic effusion, but owing to the rigid chest walls the diagnosis must be made by percussion and auscultation in different positions. The interpretation of heart murmurs occasionally gives some trouble if two or more valves are affected, especially if in addition there is an aortic bruit. Combined val-

vular defects are the rule in old age and when the rhythm is irregular it is often impossible to make a diagnosis from the murmurs alone. It is sometimes necessary to feel the carotid pulsation or the apex beat to determine whether a murmur is systolic or diastolic. In aortic stenosis the murmur may be loud enough to mask the less audible systolic murmurs of mitral regurgitation and dilatation of the arch of the aorta, while in aortic and mitral regurgitation and in aortic obstruction with dilated aorta—the most frequent combination of valvular lesions—systolic and diastolic murmurs are heard all over the chest and the diagnosis must be made by accompanying symptoms and signs. Notwithstanding the vastly inferior methods of diagnosis of abdominal disorders as compared with the methods applicable to thoracic diseases, errors in diagnosis are less liable to happen in the former class of cases. The principal sources of error in abdominal disease are absence of pain in usually painful diseases especially inflammations, abnormal position of organs or tissues, symptoms apparently connected with other organs than the one diseased, symptoms referable to a diseased organ but differing from the ordinary symptoms of the suspected disease, and manifestations of senility simulating a disease. Some of these sources of error have already been discussed. An example of symptoms referable to other than the diseased organ is seen in the asthma and vertigo frequently associated with acute gastritis, and sometimes more pronounced than the gastric symptoms. The absence of prominent symptoms of a disease occurs more frequently than the presence of exceptional symptoms. Postmortem examinations frequently reveal lesions that gave no symptoms during life, even of such diseases which give pronounced symptoms and signs when occurring in maturity. Gastric ulcers have been found after death in cases where there had been no pain, vomiting or hyperacidity during life. Vomiting is, however, frequently absent in old age in diseases in which it is a prominent symptom in maturity. Diarrhea is comparatively infrequent in the aged and in almost every case can be traced to some fault in the food. When it occurs in connection with other diseases it has little or no diagnostic value. Constipation, when the only symptom, is generally due to diminished peristalsis and has no diagnostic value, except as an expression of the physiological senile changes in the intestines. When associated with other symptoms not

due to the constipation, its significance is uncertain. (This and diarrhea will be discussed fully in the article on senile changes in the intestines.) In many cases of abdominal disease the etiology and history of the case will give more information than the symptoms and signs. On the whole the diagnosis of this class of diseases is not difficult if we remember the senile changes and eliminate their normal manifestations. Far more difficult is the diagnosis of diseases of the nervous system owing to the diverse character of the senile changes in the organs, and their manifestations. It is often a question of personal opinion whether the functional changes are normal or abnormal, physiological or pathological. The difficulty is increased through the resemblance of some of the altered functions to the impaired functions of certain disease conditions. The senile gait and senile tremor may resemble the gait and tremor of paralysis agitans, the senile dementia of cerebral atrophy is like the dementia of cerebral softening and the dementia following melancholia; the changed reflexes in old age suggest various nervous diseases. The altered reflexes and weakened power of coordination that we frequently find are symptoms of well-defined diseases, yet postmortem examinations may fail to show the lesions associated with such diseases. Notwithstanding these difficulties the practical elimination in old age of tabes dorsalis and diseases involving increased functional activity simplifies the diagnosis. Of the general neuroses senile tremor and paralysis agitans are most frequent, and neurasthenia is sometimes seen. Vertigo occurs quite frequently in old age and is almost always due to cerebral arteriosclerosis. Of the psychoses, melancholia and hypochondria are frequent, occasionally there is amentia or paranoia, rarely mania. There is little difficulty in their diagnosis. Dementia is the usual outcome of the psychoses of old age and the termination of senile atrophy and cerebral softening. A temporary dementia may follow apoplexy. Arteriosclerosis of the cerebral vessels is the most frequent cause of mental impairment and the same disease in the vessels of the cord is responsible for many of the diseases of the cord and spinal nerves. Meningeal diseases are rare in old age and when they do occur at that time of life they are almost always secondary. Other cerebral diseases, such as anemia, hyperemia, hemorrhage, embolus and thrombus and the diseases resulting from them, are generally



The "worm-eaten" brain. A rare and extreme form of senile degeneration. (Williams *Medical Record*, Nov. 23, 1912.)

traceable to atheroma and present no serious difficulty in their diagnosis as they do not differ from the same diseases occurring in maturity. Some writers describe a senile paraplegia as a distinctive disease, but there is no unanimity in their description of the disease and they agree only on one symptom, a progressive weakness of the lower limbs. As this may be due to various conditions of the brain and cord and to the physiological changes caused by ageing—perhaps to the simple waste of muscle—the term senile paraplegia will be used to denote the one symptom and not a well-defined disease. The most frequent spinal disease in old age is myelitis, although there are frequently symptoms pointing to other degenerative diseases, principally to degeneration of the lateral and posterior fibers. The principal disorders of the peripheral nerves are neuritis, neuralgia and disorders of sensation and of the special senses. In some cases it is difficult to determine whether the impairment of the special sense is due to central or to peripheral disease, especially as no change of a degenerative character has been demonstrated in the taste bulbs or in the middle ear (except waste of the drum), nor in the sensory terminals of touch. In making a diagnosis in a senile case, we must determine to what extent the symptoms are modified by the mental state of the individual. Weakness and the fear of falling may produce a gait similar to the gait of spastic paralysis. Mental dulness may produce lessened appreciation of pain, and on the other hand fear of pain may cause excessive sensitiveness, hyperesthesia and even paresthesia. Whenever we must depend upon the patient's intelligence for diagnostic information, we must endeavor to secure corroboration of the patient's statements. Incidental complications, *i.e.*, those due to the senile disease, and accidental complications, those not due to or connected with the primary disease, occur frequently in the course of senile diseases. In maturity such secondary diseases are often preventable and generally curable. In senility they are rarely either avoidable or curable, as they are caused by the efforts of co-related organs to maintain harmonious relations with the diseased organs, such efforts increasing or perverting the functions of the secondary organs and hastening their own degeneration. In the pneumonia of maturity, for example, the action of the heart is increased in force and rapidity, thereby increasing pulmonary circulation in the unaffected part of the lungs, while

the increased respiration serves to oxygenate the increased amount of blood sent by the heart, thereby maintaining the circulation of properly oxygenated blood throughout the system. If the heart is in good condition it can keep up this rapid pace for days without impairment. In pneumonia in old age the increased activity of the heart, which is already working to the limit of its capacity, rapidly exhausts the organ. Death from disease in old age is rarely due to the primary disease but to the inevitable secondary involvement of vital organs or to general physical exhaustion. In making a prognosis we must consider not only the disease itself, but the capacity of the co-related organs and to what extent they can stand further strain upon them. In the treatment of diseases the first as well as the ultimate aim of the physician should be to prevent the immediate cause of death. If there is a persisting cause of the disease which can be reached and removed, that should receive attention before any treatment itself is instituted. In most cases, however, the cause even if persisting cannot be removed and we must treat the results. In the great majority of cases the immediate cause of death, *i.e.*, the determining factor which causes death, is not the disease but either general exhaustion or exhaustion with paralysis of the heart. There are other immediate causes of death, such as paralysis of the brain, shock, asphyxia, etc., but general asthenia and heart failure are the most prevalent and the danger that one or the other may set in is present in almost every senile disease. The prevention of these two dangers must therefore engage the physician's attention from the beginning of every disease in old age. Even in diseases like apoplexy, cerebral embolism and various toxemias which paralyze the brain, cases occur which are prolonged, and secondary conditions arise which may end in exhaustion or heart failure. These dangers should be guarded against as soon as the secondary conditions appear.

A serious difficulty in the treatment of diseases of old age is the uncertainty of the action of drugs upon the senile organism. We know little of the physiological action of drugs upon normally degenerating tissue and we know virtually nothing of the therapeutic action of drugs upon diseased degenerating tissue. Drugs which are almost specifics in certain diseases in maturity may be ineffectual in similar conditions in senility. Assimilation is

changed and drug activity is slower and prolonged and we consequently get the effects that smaller doses produce. Secondary effects are sometimes more pronounced than the primary ones and thus we may get unexpected results. In some cases the etiological factors influence the action of drugs. In arteriosclerosis due to alcoholism, lead, syphilis, gout, nephritis and other diseases, the cure of the disease is accompanied by an improvement in the condition of the vessels. In such cases the iodides will cure arteriosclerosis. If, however, the disease of the arteries is not secondary but is merely a simple senile degeneration, the iodides have no action other than the physiological effect of the drug upon the organism. Drugs that have a beneficial effect upon the kidney of interstitial nephritis have no effect upon the senile contracted kidney, although the two resemble each other and the senile kidney may present an albuminous urine. This may be used as an argument in favor of the statement that the senile degenerations are essentially different from the diseases which present the same morphological features in maturity, that for example the arteriosclerosis following syphilis is a different disease from the arteriosclerosis which appears as the physiological senile change. In some cases where the incidental effects of drugs are more pronounced than the primary effect, the secondary effect may destroy the primary effect or produce other deleterious results. This is well seen in using digitalis as a heart tonic in cases where there is arteriosclerosis. The drug acts primarily as a heart tonic, increasing the force of the contractions. The secondary effect is vasoconstriction whereby the lumen of the vessels is diminished. Fortunately digitalis in powder or tincture acts slowly, but if the active principle is used hypodermically the action is more rapid, and atheromatous cerebral vessels are contracted and at the same time they are subjected to the increased pressure exerted by the heart. Being unable to stand the strain they rupture and apoplexy is the result. Some drugs which are readily absorbed in maturity are absorbed so slowly in senility as to be virtually inert. This is the case with cinchona and other tannin-bearing drugs. The same sometimes happens with gelatine, and gelatine-coated pills and capsules may pass through the stomach unchanged. Owing to the generally slower assimilation and the constipation of old age cumulative effects are more frequent at that

period of life. This is especially true of opium and belladonna which lessen peristalsis in the already weakened intestines. In combining drugs to overcome undesirable by-effects the corrective may itself have undesirable secondary effects. This is seen in the popular aloin, strychnine and belladonna pill in which the belladonna is given to overcome the griping effect of the aloin, by allaying the peristalsis, to produce which we give the aloin. Incidentally the belladonna lessens the intestinal secretions making it still more undesirable in senile constipation. The usual combination of morphine and atropia is irrational and dangerous in old age as it gives a false sense of security in cases where morphine action is desired. Morphine beside its primary analgesic effect paralyzes the respiratory centers and to prevent this the atropia is added. But morphine acts more rapidly than atropia and in the aged where these centers are already weakened, the morphine may kill before the atropia has begun to act. Herein lies the great danger in giving morphine to the aged. If atropia is given a few minutes before we give the morphine or if morphine be given per os and a hypodermic of atropia is given at the same time morphine can be given in as large a dose as in maturity.

The old dictum that children and the aged cannot stand large doses does not hold good when applied to the latter except in a few drugs. Many drugs can and must be given in larger doses in old age to be effective. In senile constipation, for example, we give intestinal peristaltic stimulants, beginning with the smallest effective dose. As in time the waste and atony of the muscular fibers of the intestines proceed in the process of involution, and the peristalsis diminishes, we must gradually give larger doses of the stimulant until many times the original dose is required to have any effect. Not infrequently an initial dose of 1/8 grain of aloin must be gradually increased to 2 or 3 grains. This is not due to habituation only, for if we change the drug the new drug must be given in correspondingly large doses. The system does become habituated to a drug, especially in old age, but this can be readily overcome by an occasional change.

As weakened functional activity and secondary effects resulting therefrom are the most prevalent of the senile ailments, tonics and stimulants are the drugs mostly used in senile cases,

and as functional weakness increases increased doses of the drugs must be given. Sedatives and hypnotics are rarely required in old age although they are apparently often indicated. When they are employed they should be given in the smallest effective dose, reduced after the initial dose and stopped entirely as soon as the desired effect is obtained. This does not apply to cases where the full effect is to be derived from a single dose, as when giving an analgesic. In such cases a single full dose, to be given with proper precautions for avoiding the incidental effects of the drug, is better than repeated small doses. It is often impossible to decide whether the pain from which the patient complains is real or whether the fear of pain creates the impression of pain or produces an oversensitiveness that exaggerates simple tenderness into pain. If the disease is usually painful there will probably be real pain and morphine is indicated. If it be merely oversensitiveness a placebo, preferably one containing aloes, quinine or some other disagreeable drug, should be given. In many cases the patient will rather stand the pain than take the drug and he will often declare that the pain is bearable or has entirely disappeared under such treatment. The aged frequently complain that they cannot sleep at night and the physician is tempted to give a hypnotic. On close questioning in these cases it will be found that while the aged patient cannot sleep for more than a few hours at night he takes frequent naps during the day and the total amount of his sleep in naps, dozes and sound sleep may be from ten to fifteen hours out of the twenty-four. These cases are hard to handle as the patient does not realize that his naps sometimes last for hours, that the little doze after reading the papers, etc. (really due to brain fag), is sleep, and that he sleeps so much in short stretches during the day that the system does not require more than a few hours sleep at night. Where there is real insomnia it is better to try hot baths, hot drinks, suggestion and other non-medicinal measures before resorting to hypnotics. Chloral is useless in small doses and dangerous in large doses on account of its depressing effect upon the heart. Veronal is perhaps the safest hypnotic in old age and if the insomnia is due to mental agitation —a frequent cause in the aged—veronal and monobromated camphor is a safe and effective combination. The bromides are occasionally required to allay nervous excitability. The

sodium salt is preferable to the potassium salt as the former contains 10 per cent. more of the bromine element and much less of the alkali than the latter and is besides not as irritating to the stomach. The chronic nervous diseases of maturity seldom reach old age and rarely originate in old age. Paralysis agitans, the one which occurs most frequently in the aged, is not influenced by the bromides, while senile tremor is aggravated by this class of drugs.

Drug action is influenced by the mode of administration. Drugs should never be given in the form of gelatine-coated pills or capsules. Salts given in solution are absorbed quickly but if given in powder form, their action depends upon their solubility. Hours and sometimes days pass before the action of insoluble salts like calomel and bismuth is recognized. Drugs like arsenic, phosphorus, etc., which may produce local irritation in the stomach, should always be given in solution well diluted. Where there is danger of cumulative effects care should be taken to secure free evacuation of the bowels by active cathartics. The gastric and intestinal ferments have little effect in old age but predigested food is rapidly absorbed. Drugs used for local absorption by inunction must be combined with an animal base, either lanoline, lard or sweet butter. Vegetable fats and oils are absorbed with difficulty—and mineral bases are not absorbed at all by the dry skin in old age. The same applies to liniments. They may produce local irritation through friction but an animal oil or alcohol is necessary if we want to secure the absorption of the drug.

Hydrotherapy has a wide range of application in senile cases. The aged object to the inconvenience connected with entering a bath especially if their joints are stiff. They cannot stand the shock of a cold bath and even an ice bag may give a dangerous shock. Neither can they stand the depletion produced by excessive diaphoresis. Tepid and warm baths and packs act well in every case where a temporary sedative action is desired, and a tepid bath followed by friction acts as a stimulant. A warm bath followed by inunction will sometimes relieve the stiffness of joints due to the senile changes and occasionally the stiffness found in various forms of arthritis.

Electrotherapy has not been sufficiently studied in the aged to make a positive statement as to its value. Where there is a

partial electrical reaction of degeneration the faradic current will produce a temporary stimulation of nerves and muscles, but if the reaction of degeneration is complete the faradic current has no effect. Mechanotherapy has some application in senile cases as massage, friction and passive exercise. It should be remembered that excessive activity in muscles hastens their degeneration and the increased activity of the heart in forced active exercise may cause loss of compensation and rapid exhaustion. The condition of the heart must be the guide in the application of mechanotherapeutics in the aged.

Serotherapy has not been sufficiently employed in the aged to determine its value. The diphtheria antitoxin has a more profound systemic action on the aged than in childhood and the danger from anaphylaxis is apparently greater. This last was probably the cause of the unfortunate results obtained from the use of Brown-Sequard's testicular extract.

Treatment at mineral springs is much more in vogue in Europe than in America. Mineral waters generally are contraindicated in the aged and there are but few diseases in which the benefits derived will compensate for their disadvantages. The only diseases in the aged which show greater improvement from a course of treatment at springs than from home measures are cholelithiasis, diabetes and gout. It is probable that the strict regimen enforced at the European springs contributes as much to the result as the waters themselves, because the same regimen at home with the bottled waters does not produce the same results. The psychic influence is absent at home and even at American springs where the patient can keep in touch with his home and business and thus keep up the cares and worries which frequently contribute to the disease.

In the treatment of diseases the author mentions only such drugs and measures as he has found to be of service, omitting the host of drugs and measures recommended by other writers.

The senile changes bring about vicious circles which increase in number and size until every organ and tissue and every function is involved. In atheroma the weakened elastic fibers diminish the elasticity of the vessels and the heart must send blood with greater force through these vessels to maintain the circulation. This puts the fibers still further on the stretch, weakening

them more and more. The weakened muscular fibers of the colon permit dilatation of that part of the intestines and feces collect in the pouch thus formed, distending the pouch, stretching the remaining fibers and further weakening them. Similar vicious circles are formed in the stomach and bladder. The dilated stomach permits the accumulation of food which further weakens its walls, the dilatation is increased through this accumulation of food and gas and this in turn impairs the digestive power of the organ.

The dilated atonic bladder holds urine through its inability to void it; this increases the dilatation and permits more urine to collect in the larger saccules thus formed, the sacs stretching the muscular fibers and weakening them more and more. Owing to the atrophy of the lung and waste of the interalveolar septa, the aerating surface of the lungs is diminished and the blood is improperly oxygenated. The capacity of the blood to carry nutrition to the organs is thereby impaired and this includes the lungs where the increasing atrophy further diminishes the aerating surface. The most pernicious of the vicious circles is formed in the heart after the limit of compensatory hypertrophy is reached. The heart is now no longer able to overcome the impairment caused by diminished expansibility of the arteries, dilated veins and weakened valves, its tonicity is lessened and it sends blood with less force through the system. The circulation is slowed and weakened and the elimination of waste and the supply of nutrition is slower and thus the nutrition of the heart itself is impaired. This further weakens the organ and still further weakens the circulation. Retardation of the pulmonary circulation causes an accumulation of blood in the right heart, producing dilatation and under the combined influences of stretching of the cardiac walls, insufficient nutrition and excessive work to empty overfilled cavities, the heart rapidly degenerates, it becomes exhausted or paralyzed. It requires careful discrimination to separate the manifestations of physiological senile changes from the symptoms of disease. The treatment of the diseases in the aged is still mainly empirical; every case requires individual attention and routine measures based upon the same conditions as found in maturity are certain to lead to disaster. We must look upon senility apart from maturity and its diseases, as sui generis senile diseases.

CLASSIFICATION OF DISEASES IN OLD AGE

There is probably no other branch of science in which nomenclature and classification are as imperfect as in medicine. Some medical terms indicate the pathological condition, as chronic parenchymatous nephritis, or acute follicular tonsillitis; some point to the etiology, as sunstroke, hay fever, etc.; some are purely symptomatic, as neuralgia, tachycardia, hematemesis; some are generic and are applied to several pathological conditions which resemble each other in one or more symptoms or in the location of symptoms, as rheumatism, pneumonia; some bear the name of the physician who first described the symptoms or investigated the disease, as Bright's Disease, Addison's Disease, Bell's Paralysis; while some terms do not refer to either the etiology, pathology or symptoms present.

In our nosology there is neither order nor system. One author follows an alphabetical arrangement beginning with abortion and ending with yellow fever. Older writers generally classify diseases according to the organs or system of organs in which they occur, as diseases of the circulatory system, digestive system, etc. Recent authors use an etiological and regional classification separating infectious and parasitic diseases from the others and dividing the latter according to the system to which the affected organ belongs. A more recent classification is devised according to the initial cause and divides diseases into physical, chemical, animate, mental and nutritional diseases. A revolutionary revision of our nomenclature is necessary before we can place upon a scientific basis medical terms and the classification of diseases, and until this is accomplished every classification must be imperfect.

The basis of the classification employed in this book is the relation of the pathological condition to the senile organism. It divides the diseases found in the aged into five groups as follows:

(1) Primary senile diseases, *i.e.*, diseases in which there is an increase, decrease or perversion of the ordinary senile anatomical or physiological changes.

(2) Secondary senile diseases, *i.e.*, diseases which result from the senile changes.

(3) Modified diseases of old age, *i.e.*, diseases which, when occurring in old age are modified by the senile conditions, or present features not found in maturity.

(4) Preferential diseases of old age, *i.e.*, diseases which occur most frequently in advanced life.

(5) Diseases uninfluenced by age or are rare in old age. Strictly speaking, every disease is influenced by age but the diseases of the fifth group are those which do not differ materially in etiology, pathology or symptoms from the same diseases in maturity.

The first group includes diseases which present abnormalities in the normal process of involution. As we have, however, no standard of the normal senile conditions and no means of establishing a norm, it will be necessary to include the ordinary senile degenerations under this heading as nearly all produce discomfort or give rise to secondary pathological conditions. We must remember that even slight changes may cause profound functional manifestations and, on the other hand, there may be extensive anatomical changes in organs and tissues, without disturbing their harmonious relations with allied organs and tissues or producing symptoms of disease. The true senile diseases may be primary, *i.e.*, the cells degenerate through some property in the cells themselves, such as has been suggested in the theory of tissue-cell evolution, or they may be secondary to arteriosclerosis and then due to malnutrition. These are all included under the first group. Changes identical with senile changes may be found as the result of other etiological factors. Arteriosclerosis may be due to syphilis and other toxemias, to excessive food during prolonged inactivity, to cardiac disease, etc. It is possible that future research may disclose some intrinsic difference between the tissues degenerating through the normal process of involution and those degenerating from disease. That there is some difference is evident from the functional differences and from a difference in the action of drugs.

Diseases of the first group are organic or functional, the functional diseases presenting functional perversions for which no histological change can be found. Included in the functional diseases are senile tremor, senile impotence, senile pruritus, senile cachexia, and true senile dementia which is a symptom of cerebral atrophy and degeneration.

Many diseases belong to two or more groups. These will be described in the group most closely allied to the senile state, as chronic endocarditis which may be primary, secondary or

modified and which occurs most frequently after middle age, and is placed in the first group under degenerations of the heart. Senile emphysema belongs to the first group while ordinary emphysema which is rare in the aged and does not differ from the same disease in earlier life, is omitted. Senile non-infectious pneumonia belongs to the second group, while infectious pneumonia, whether localized or diffused, belongs to the fifth group. In order to preserve continuity of description it was found advisable in some cases to describe a disease belonging to one group with a disease of another group. Doubtful etiology may have caused improper grouping. Arrhythmia which is probably due to some disturbance of the vagus and would therefore belong to diseases of the nerves, is placed in the second group under cardiac neuroses and angina pectoris is placed in the same class, although it is often a symptom of coronary arteriosclerosis which belongs under primary senile diseases. Senile tremor is placed in the first group on the assumption that it is a symptom of general debility of the aged and due to cerebrospinal degeneration.

Where the etiology and pathology of a functional disorder is unknown or uncertain it is placed with diseases of a like character which can be classified. Other diseases with obscure etiology and pathology are placed in the fourth group if they occur frequently in the aged, or else in the fifth group if they do not fit under any other head.

This classification must be revised as our knowledge of the pathogenesis of diseases, like gout, diabetes, cancer, pernicious anemia, etc., increases.

Parasitic diseases, rare tropical diseases and diseases which in the aged do not differ from the diseases of maturity have generally been omitted.

PRIMARY SENILE DISEASES

SENILE CACHEXIA

Senile Debility

This term is employed to cover the vitiated condition of the senile organism. It includes the lowered functional activity and capacity and the obvious manifestations of ageing, which form the tout ensemble of senile debility.

Pathology. The Blood.—While neither chemical nor microscopic examination of the blood of the aged has revealed any abnormal constituent or any marked disproportion of normal constituents as compared with the blood of younger individuals, there is undoubtedly some change in the character of the blood of the aged. It has a high percentage of hemoglobin in spite of its readiness to part with it to form pigment deposits in the areas of degeneration and of passive hyperemia. It has a tendency to hold the products of defective metabolism thereby giving rise to the diseases of metabolism. Its nutritive value is lowered, as its ability to carry nutrition to the organs and waste from the organs is diminished. In twelve out of thirteen examinations reported by Grawitz, the leucocytes numbered between 4000 and 8000, and the red cells between 4,470,000 and 5,300,000, the hemoglobin percentage was between 90 and 110 and specific gravity between 1051 and 1060. Notwithstanding the profound anatomical change in the senile spleen and in the character of the bone marrow, the number of cells are not reduced. The high specific gravity is due to a diminution of the watery element and the blood of the aged is consequently more viscid than in maturity. This favors coagulability with slowed current and production of thrombi and emboli. With the same proportion of water as in earlier life the other constituents would be proportionately reduced and there would be an anemia with deficient cell elements and salts. The proportion of chloride of sodium is less in the blood of the aged and there is an increase in lime but these variations are slight. (Other anatomical and physiological changes that contribute to the general condition are described under anatomical and physiological changes in old age.) The sallowness of the aged is not due to the condition of the blood but to deficient surface circulation and consequent changes in the integument. In many cases of senile debility the anatomical and physiological changes are slight and the condition can be traced to psychic causes.

Etiology.—The underlying causes of senile cachexia are the underlying causes of ageing. The obvious manifestations are due mostly to the anatomical and physiological changes. There are, however, some etiological factors that deserve special consideration. There is a remarkable similarity between the cachexia of old age and the cachexia of unsanitary life. In the latter there is generally insufficient food, in the former there

is impaired assimilation of food producing the same effect. The aged do not get sufficient air into their lungs to completely oxygenate the blood owing to anatomical changes in the lungs and chest wall. Those living unsanitary lives do not get sufficient pure air into their lungs owing to unwholesome surroundings. The effect of insufficient sunshine upon the latter has its counterpart in the effect of sunshine upon the weather-beaten skin. In both cases there are sallowness, weakened vital functions, lessened resistance to disease, slow and incomplete recuperation, yet in both the blood count is normal and the cells show no abnormalities. It is probable that sunlight itself or its absence is a factor in ageing, as those who are deprived of sunlight, like miners, persons working in cellars or who work at night, are usually sallow or pale and age rapidly, while the withdrawal from night work has a rejuvenating effect.

The general physical weakness that accompanies ageing is due to the anatomical and histological changes in the joints, the waste and atony of muscle and lessened innervation. In the muscles the contractile power is diminished, and the responses to stimuli and to the will are slower and less active, fatigue sets in more rapidly, is more profound, and recovery takes longer. The joints become stiffened; coordination is more difficult and it often requires a conscious effort or impulse to bring about coordinate movements that have usually been performed unconsciously.

In addition to these senile changes involved in the production of senile debility, there is always a psychic factor which may be more pronounced than the senile changes. This psychic factor may be causative or resultant, aiding in the production of senile debility or arising from a recognition of such debility, but in either case it tends to exaggerate the objective and subjective manifestations of this condition. True physical debility caused by the anatomical changes in the bones, joints, muscles and motor nerves, is progressive, the extent of the weakness depending upon the extent of the senile changes, and temporary forced stimulation is followed by more rapid degeneration. In many cases, however, the debility is apparently greater than the anatomical changes would warrant, while there is a profound mental depression without marked mental impairment. In these cases the debility bears a relation to the mental

attitude and little or no relation to the physical condition of the individual. In every case of senile debility physical and psychic factors are involved, the latter playing but an insignificant part in some cases, while in others it may be the main etiological factor.

Symptoms.—Obvious manifestations of senile debility are described in the chapter on the Senile State. Aside from the changes in the skin and hair, and waste of tissue, the most pronounced manifestation is the posture and gait of the aged. Owing to anatomical changes in the spinal column and chest walls there is an exaggerated dorsal curvature and flattening or retraction of the anterior surface of the chest, the weakened muscles of the back and neck allow the body to sink and the head to fall forward; the shoulders droop, the arms hang, and the lower limbs are bent at the hips and knees to maintain equilibrium. This characteristic senile stoop does not appear in any disease but it may be simulated by the slouch of psychic pseudo-senile debility. It is necessary to distinguish between the senile stoop and the senile slouch—the former due to the anatomical changes and coming on slowly and late; the latter due to psychic causes and coming on rapidly and early.

The most natural position of an individual is the one involving the least physical effort, namely, one permitting complete relaxation of the muscles. The ordinary position with head up, shoulders thrown back, chest out, the individual standing as erect as possible, is the result of effort which long continued finally becomes a habit. The child is taught to sit straight and to stand straight yet there is always the tendency to relapse into slouching position. This tendency overcomes the habit during sleep, under depressing emotion, and in some persons in whom the effort to maintain the erect posture has not become a fixed habit. The habit is later maintained by a sense of pride in one's appearance, the erect bearing being more pleasing to the eye, the individual being thereby better able to secure public recognition and approval. When an aged person begins to feel the infirmities that come with advancing years, the labored breathing upon slight physical effort, the fatigue that sets in rapidly, the stiffening of the joints and the fact that the usual labors become more difficult—he then realizes that he is on the downward journey of life. To some this comes as a shock, to others as

the realization of a long anticipated misfortune. It produces a mental depression, which is sometimes so profound that ambition is lost, there is no longer any pride in appearance and the mind is centered upon life itself. Some fear that they have not provided sufficiently for their declining years, others that they may become a burden upon those who might wish to be relieved of this burden. In some cases the loss of sexual virility will produce this mental depression. Whatever the cause may be, the loss of pride in the carriage or bearing of the individual brings about the natural tendency to slouch and the individual assumes this position. Worry will hasten the appearance of age and in a short time the ageing individual presents the general appearance of old age and senile debility. We frequently find that an improvement in the mental condition is followed by restoration of physical vigor and it is generally noted that decrepit persons lose the appearance of decrepitude and gain in physical strength upon their admission to a home or asylum where they are free from worry. In almost every case where senile debility occurs early and proceeds rapidly the psychic factor is the main cause.

The impairment of the special senses, mental impairment, intensified emotions, especially fear, minor physical defects, as broken-down arches, hypersensitiveness, etc., must all be included in the conception of senile debility as they all increase the helplessness of the individual. These are wilfully exaggerated in pseudo-senile debility but appear none the less real to the patient. It is difficult to draw a sharp line between this condition and senile neurasthenia. The neurasthenic generally maintains his pride in appearance and overcomes the tendency to slouch or he may lapse into a slouch from which he can be roused with little effort, while it often requires all the skill and tact of the physician to rouse the other even temporarily.

Senile debility is sometimes complicated by senile tremor and senile dementia. The tremor is probably due to degeneration of the spinal cord and will be treated under that head. It does not appear in debility of psychic origin except when acquired through imitation. A slow, progressive senile debility and dementia occur normally after the senile climacteric and may then simulate general paresis. The latter disease occurs earlier in life, there is usually a specific history, a history of convulsions,

delusions of grandeur, rapid mental and physical decay with periods of temporary improvement. These features are absent in senile debility with dementia. There are several pathological conditions marked by tissue waste and debility without other pronounced symptoms of disease. Schoenlein's senile marasmus—an atrophy of the stomach and intestines—may simulate true senile debility—or may occur with the latter. In Schoenlein's disease there is an excessive amount of feces and lientery, while the characteristic stoop is absent unless senile debility is present.

Carcinoma, tuberculosis, marasmus, etc., produce waste of tissue and debility, but in these cases the rapid emaciation attracts the physician's attention and he looks for a well-defined disease. Tuberculosis especially is liable to be mistaken for senile debility and its true nature may be overlooked until the disease is far advanced. Rapid emaciation with debility and without other marked symptoms or with an afternoon rise in temperature points to tuberculosis.

Treatment.—The treatment of senile debility includes medical, psychic and hygienic measures.

The medicinal measures have for their object, (1) the functional stimulation of muscles and nerves, (2) the relief of the stiffness of the joints, (3) improvement of the mental attitude, (4) relief of minor ailments associated with senile debility, (5) general tonic treatment.

Phosphorus, strychnine, and arsenic meet the first, third, and fifth of these indications. Arsenic increases bodily vigor, stimulates the appetite, favors constructive metabolism and improves the general physical condition of the individual. It is the most valuable tonic for the aged, but it is a treacherous drug, the limit of tolerance being sometimes reached in two or three months and at other times two or three doses given in twelve-hour intervals to the same person will produce a cumulative toxic effect of the combined dose. Strychnine is a powerful nerve stimulant, having, however, the serious drawback that it increases heart action as well. If there is cardiac hypertrophy —the usual condition of the heart before decompensation—it is contraindicated. When there is no contraindication to its use it can be combined with arsenic as strychnine arsenate in doses of 1/100 grain. The dose of the arsenic in this salt is small and

there is no danger of rapid toxic effect. If there is an objection to strychnine, the arsenic should be given alone or with phosphorus, in doses of 1/40–1/20 grain of the arsenic trioxide or six minims of Fowler's solution twice daily. As soon as cramps or pain in the stomach, a distaste for food, or swelling under the eyelids appear, the drug must be stopped. Phosphorus is a mental stimulant, nerve tonic and aphrodisiac which has no cumulative effect, and no reaction; its action is prolonged and it can be discontinued without detrimental effect. It increases mental activity and produces a sense of well being, rousing frequently a desire for increased physical exercise. It can be given in doses of 1/100 grain of the ordinary phosphorus, or 2 grains of the amorphous, non-toxic red phosphorus. Lecithin, notwithstanding its organic phosphorus content, does not produce the same effect as the inorganic drug.

Phosphorus should be given until there is a noticeable improvement in the patient's mental attitude. Whenever mental depression appears again its use must be resumed in an increased dose.

Opium and its preparations are active cerebral stimulants in small doses, but the effect soon wears off and there is danger of habituation. They are not mental restoratives, for the reasoning power acts vicariously; ideas are more florid, the imagination is stimulated, but memory is not improved, as neither the receptive nor retentive power is strengthened. Cocaine is a cerebral stimulant and produces a sense of well being but it is always dangerous in the aged especially so in the arteriosclerotic. Coffee or caffeine can be used as a general stimulant without danger.

The treatment for stiffening of the joints is given in the chapter on Arthrosclerosis Senilis and the treatment of the minor ailments is given under their various headings.

Psychic measures are most important in pseudo-senile debility and are of some service in the true senile debility. The old soldier hobbling along on Decoration Day makes a firmer step, walks erect and becomes spry and lively as he passes the reviewing stand. Flattery has a more permanent rejuvenating effect, especially flattery from a young person of the opposite sex. Association with younger persons on the plane of companionship and especially marriage with a younger person will

do more to dispel the feeling of mental and physical debility than any medical measures. The ancient Romeo who goes courting becomes young in feelings and forces himself to both actions and looks to correspond with his mental attitude. Where the psychic factor causing the general weakness was worry, relief from this worry will relieve the debility. This accounts for the improvement of aged persons immediately upon a change of environment—interesting sights that do not confuse or fatigue, old familiar, lively airs, the pursuit of a harmless hobby; anything that will tend to divert the mind from the body and from death will have a beneficial effect upon them. The benefit derived from the freedom from worry upon entering a home for the aged, is soon dissipated when the aged person finds his associates complaining of their petty ills and when he sees these associates dying. The aged person should have a pleasant young companion, preferably of the opposite sex, constantly around him.

The hygienic treatment will be given in the chapter on Hygiene.

SENILE ARTERIOSCLEROSIS

Arteriosclerosis is the most frequent and in its consequences the most important of all senile degenerations. Faulty nomenclature and a failure to differentiate between different forms of arterial degeneration are responsible for the many misconceptions concerning this condition which in its milder form is natural and normal in old age.

The terms arteriosclerosis, atherosclerosis, atheroma, arteriocapillary fibrosis, arterial sclerosis, atheromatosis, arteritis deformans, endarteritis nodosa, endarteritis deformans, periarteritis, have all been used interchangeably or to designate one form or another of arterial degeneration, thereby creating confusion. Bishop introduced the term cardiovascular disease to cover a clinical syndrome including disease of the heart and of the blood-vessels, arteriosclerosis and the co-related conditions of autointoxication, neurasthenia, kidney degeneration, etc.

The term is unfortunate since it includes many conditions which differ in association, pathology and symptoms, hence it does not represent a definite entity.

The term senile arteriosclerosis is applied to a form of arterial degeneration which is part of the process of involution, not due to antecedent disease, is progressive and incurable. A pure senile arterial degeneration uninfluenced by any other factor than the underlying cause of ageing is hardly conceivable. It will therefore be necessary to describe other forms in order to understand the ordinary degeneration found in the aged. Arteriosclerosis is divided, (1) as to extent, into circumscribed and diffuse or general, the former involving one or more circumscribed areas, the latter involving to some extent most or all of the arteries of the body; (2) as to location, aortic, cerebral, coronary, radial, etc., depending upon the vessel involved; (3) as to etiology, physiological as occurs in the normal process of involution, presenile when the process is normal but hastened, and pathological when due to disease; (4) as to pathology, inflammatory when beginning as an endarteritis, mechanical when beginning by loss of tonicity of the muscular coat, and nutritional when due to interference with the nutrition of the vessel through inflammation or blocking of the vasa vasorum; (5) as to prognosis, temporary and permanent, the former curable, the latter incurable; (6) primary or secondary, the latter when following and due to another disease.

Etiology.—The basic cause of physiological or senile arteriosclerosis is the basic cause or causes of ageing. Any of the fundamental causes of senile involution, whether ascribing the initial changes to the blood, to the blood-vessels or to the cells can be made to fit the etiology of senile arteriosclerosis.

The prevalent view of German physicians favors Thoma's histo-mechanical theory and loss of tonicity of the muscle fibers. In France and America Metchnikoff's autointoxication theory with endothelial irritation and inflammation is favored. A more recent theory ascribes the basic cause to hyperactivity of the adrenals whereby a contraction of the arterioles is produced and consequently the vasa vasorum receive a diminished supply of blood and the larger vessels are insufficiently nourished. While the senile changes in the blood-vessels can be explained by the cell-evolution theory, it has not been verified and it is presented here as a possible cause, acting alone or in combination with other causes. The main objection to Thoma's theory is the fact that in most cases endarteritis or degeneration due to

insufficient nutrition occurs before the appearance of muscle atonicity, as evidenced by vascular dilatation. The objections to the theory of autointoxication as the basic cause in normal degeneration are that feeding animals with sterile food causes death, that meat is supposed to be the principal source of alimentary autointoxication yet vegetarians have arteriosclerosis and the amount of meat consumed bears no relation to the extent of the degeneration, and that autointoxication goes on almost from birth. It is undoubtedly a very potent contributing factor but it cannot be accepted as the determining cause. A determining or basic cause must produce like results under like conditions and such results must always be traceable to such cause. Applying this test to the autointoxication theory we find that the absorption of the products of intestinal decomposition does not always produce arteriosclerosis nor can we trace every case of arteriosclerosis to this cause.

The discovery of Josué that the injection of adrenalin in rabbits produces arterial degeneration is the basis of the theory of adrenal hyperactivity. The adrenal secretion has a selective property upon the arterioles, contracting them thereby diminishing the blood supply to the vasa vasorum which arise from the arterioles. This would seem to indicate that the disease arising from adrenal hyperactivity has a causal relation with increased blood pressure. Adler has shown that the disease produced experimentally is not identical with arteriosclerosis in the human subject, that other substances can produce the same lesions, and that the injection of adrenalin does not always produce arterial degeneration. L. Braun, using adrenalin and amyl nitrite, produced the lesion in the aorta without increased blood pressure and a like result was obtained by the use of other substances which have little or no effect upon the blood. It is evident that the action of adrenal secretion when in excess is not due to its blood-raising property but to its toxic effect. It has been shown that nicotine will stimulate the adrenals and this has been advanced to explain the prevalence of the disease in tobacco smokers. It is, however, a question whether the effect of tobacco is due to the action of the poison upon the adrenals, or upon the vasomotor centers or to the irritant action of the nicotine upon the vessels themselves. The divergence of opinion based upon contrary results of similar experiments can

be explained only by the failure to recognize different local and general conditions. It is probable that numerous factors are engaged in the etiology of senile arteriosclerosis as well as in most pathological forms. There is the underlying cause of senile involution and contributing thereto are inherent factors as heredity and muscular activity, and acquired factors as smoking, excessive meat eating, abuse of alcohol, sexual excesses, etc. The influence of heredity cannot be satisfactorily explained and it would serve no purpose to dilate upon the many theories that have been advanced to account for hereditary influence. The effect of muscular activity has been explained by the presence in the blood of the toxins produced by muscular activity, by overstimulation of the adrenals, by irritation and inflammation of the endothelium through the greater force with which the blood is sent from the heart, and by more rapid exhaustion of the muscular fibers of the vessels.

The modus operandi of autointoxication in the production of arterial degeneration is unknown. There may be direct irritation of the arterial endothelium, irritation of the vasomotor centers, of the heart or adrenals, impairment of the pabulum, or the formation of minute emboli which partially block the arterioles. The influence of alcohol is a moot question. Edgren found 25 per cent. of his cases traceable to alcoholism, and Herz obtaining the opinions and results of observation from about 800 physicians found that over half gave alcohol as a factor. On the other hand, many physicians reported that arteriosclerosis was very rare in some regions where alcohol was habitually used even by women and children. The disease is found among total abstainers, among vegetarians and among persons who never smoke, while many aged persons with a comparatively low blood pressure and soft arteries drink, smoke and eat meat daily.

Weil's theory may be mentioned here as there are some favorable reports of a method of treatment based upon this theory. Weil found that while the normal daily elimination of CaO through the kidneys was .39 grams, in over 50 per cent. of his arteriosclerotic cases the elimination was less than .2 grams and in no case did it reach the normal notwithstanding abundant lime introduction. The conclusion is that there is an excessive lime retention with consequent disproportion of salts in the

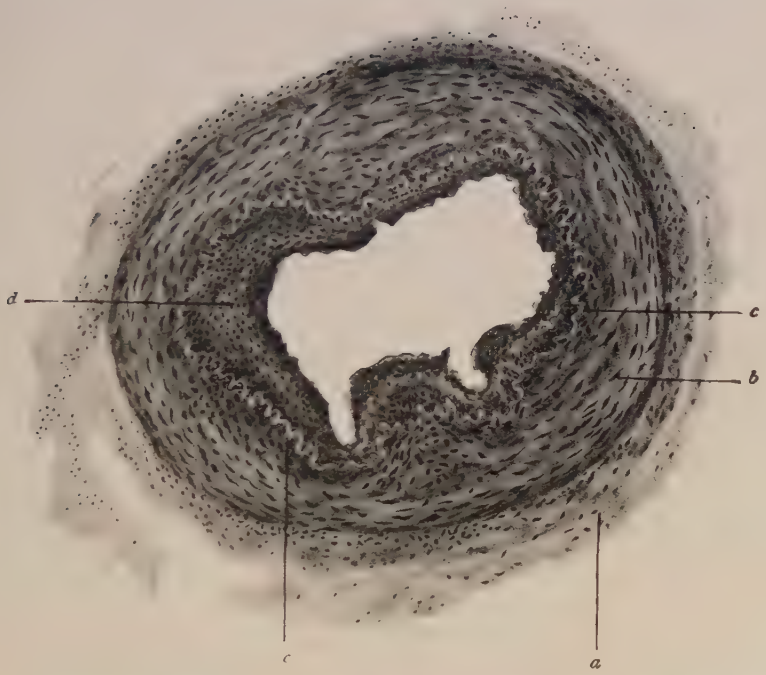
blood. Lime deposits in the vessels occur late in the disease and this theory does not explain the early degenerative process. It opens, however, a new field for speculation. As Weil's observations indicate perverted metabolism similar to the changed metabolism in gout and as late gout is marked by pathological deposits of calcareous matter in joints and other tissues while late arteriosclerosis is marked by similar deposits in the arterial walls may there not be the same metabolic disturbance underlying both diseases? The clinical picture differs according to the tissues involved. If the same perversion of metabolism—by which lime is retained in excess of the needs of the system and deposited in abnormal locations—is responsible for both gout and arteriosclerosis, the same treatment ought to be effective in both. The causes of arteriosclerosis can be placed under one of three heads: causes acting by irritating the lining membrane, or acting primarily upon the media, or acting primarily upon the vasa, vasorum either through inflammation or through diminished blood supply from contracted arterioles or increased viscosity of the blood.

Nervous, mental and emotional stress is an incidental factor acting probably through the vasomotor centers. Most other causes are toxemic, either bacteria, or endogenetic toxins, products of disturbed metabolism, or chemicals introduced from without. Their mode of action has been suggested under autointoxication.

Arteriosclerosis occurs most frequently in brain workers, in the well-to-do class, in women before the fifty-fifth year, and in men after that age.

Syphilis and lead produce a degeneration of the arterial walls so radically different from the ordinary senile arteriosclerosis that the conidtion resulting from these causes should receive a special designation such as syphilitic degeneration or lead degeneration of the arteries. They act by irritating the lining membrane.

Pathology.—The early pathology of arteriosclerosis depends upon the etiology and is determined by the location of the initial lesion. This may be in the intima, media or vasa vasorum. If the disease is due to a cause producing local endothelial irritation the earliest change is a multiplication of cells at the point of irritation. Patches of endothelium thus become thickened



Obliterative Endarteritis. (*The tissue from which drawing was made was removed from near a cancer of the face, and prepared in the laboratory of the Jefferson Medical College Hospital by Dr. Thomas Leidy Rhoads.*) 1-inch objective, 1-inch ocular. Specimen fixed in corrosive sublimate, infiltrated with paraffin, stained with hematoxylin and eosin, and mounted in balsam. *a*. Adventitia. *b*. Media. *c, c*. Elastic lamina. *d*. Irregular mass of organizing tissue superimposed on and replacing the intima. The gross specimen was hard, cord-like, but not nodular.

and soon undergo granular and fatty degeneration. The patch becomes transformed into a yellow, opaque nodular mass containing cholesterin, fatty granules and crystals, and is separated from the blood current by a thin pellicle. This atheromatous mass diminishes the lumen of the vessel. In some cases the patch contains little fat but instead a mass of dark brown granules and broken-down cells. Later, calcium deposits in the patches, first forming, in combination with the fat, calcium soaps. These break up, the calcium combines with carbonic acid and phosphoric acid radicles derived from the blood and thus the insoluble calcium carbonate and calcium phosphate are formed. The degeneration proceeds outward involving the connective tissue, which hypertrophies, and the elastic and muscular tissues, which waste. In most cases after the thickening of the endothelium, whereby the caliber of the vessel is diminished, this or some other cause interferes with the free passage of blood through the terminal vessels, the vasa vasorum receive insufficient blood supply and the main vessel is consequently insufficiently nourished. This causes waste of the highly organized muscle fiber and hyperplasia of connective tissue which requires less blood supply than the muscle structure. Still later, calcareous deposits occur in the outer coat, the deposits occurring in plaques over the patches of the inner coat or are diffused through the substance of the vessel. This is the usual course in circumscribed arteriosclerosis and forms the inflammatory type of the disease. The second or mechanical degeneration begins in the media. If there is loss of tonicity of the muscular fibers through overstretching, the vessel becomes tortuous and dilated and the current is slowed. This is followed by a compensatory thickening of the intima through proliferation of the subendothelial connective tissue. The muscular fibers having lost their elastic property become changed and they degenerate, their place being taken by fibrous connective tissue which also displaces the elastic fibers. Lime deposits occur late and are diffused. This is the usual process in senile arteriosclerosis. In small vessels the caliber may be diminished through overgrowth of the intima. In the third or nutritional type of degeneration there is more or less rapid degeneration of the media through insufficient nutrition. A diminished blood supply causes rapid waste of muscle and elastic fiber leaving minute cavities called

atheromatous abscesses and allowing small aneurysmal sacs to form. The cavities later become filled with granular and fatty débris. An increase in the connective tissue now takes place and the walls of the vessel are in spots thickened, in other spots thin and liable to rupture. Calcareous deposits occur early in the broken-down cavities.

In the condition described by the French as Aortité aigue, acute aortitis, there is an inflammatory condition of the vessel beginning at the point where the blood sent out from the left ventricle impinges upon the aortic wall, spreading downward and along the arch.

Huchard divides arteriosclerosis into four stages, an arterial stage, cardioarteriosclerotic stage, mitroarterial stage and cardiectatic stage. In the first stage, called also presclerotic stage, the toxins in the blood irritate the intima and by extension to the media cause the vessels to contract thus diminishing their caliber. The heart must now act with greater force to send the blood through the contracted vessels. In the second stage the organic changes begin in the arterial walls and heart. These are followed by nephritic changes and cerebral disturbance. In the third stage the heart becomes dilated and the blood pressure is lowered. In the last stage the secondary results of cardiac dilatation appear in the kidneys, lungs, liver, etc. There is edema of the extremities, abdomen and lungs and passive congestion of the liver.

Few cases of arteriosclerosis follow these stages as presented by Huchard. In senile cases the diminished supply of blood may produce degenerative changes in the kidneys, liver and lungs while the heart shows little alteration and no loss of compensation.

Symptoms.—The earliest subjective symptoms are referable to the organs and tissues affected by the impaired functions of the degenerated vessels. The vessels themselves give no early symptoms. When the disease is far advanced the vessel feels hard, tense, often nodular and where visible it appears tortuous. Long before these objective manifestations appear, the subjective symptoms pointing to organic disorder will call attention to the disease. The symptoms and the lesions do not always correspond, extensive areas of arteriosclerosis having been found after death which gave no evidence during life, and

in some cases symptoms usually associated with some form of arterial degeneration made life miserable yet no pathological condition to account for them could be found upon autopsy.

The earliest sign of arteriosclerosis is usually increased blood pressure but we must remember that high blood pressure and arteriosclerosis are not synonymous, and that we often find arteriosclerosis with low blood pressure.

The normal systolic blood pressure in old age can be approximately determined by adding the age to one hundred m.m. and allowing a leeway of about 5 per cent. above and below the sum of the two. Many persons have a habitual high pressure without other sign of arterial degeneration. This is found in athletes and those who do hard physical labor. The blood pressure is raised in many diseases and by many drugs, without impairment of the vessels. The diseases and drugs which raise the blood pressure may produce arteriosclerosis if their action is maintained long enough. Indicanuria is not a symptom of arteriosclerosis but it is frequently found in that disease and whenever it is found in an aged individual it is almost certain to be accompanied by high blood pressure and other signs of arterial degeneration. Bishop has shown that in neurasthenia there is usually a low blood pressure though arteriosclerosis may be present. It is probable that the increased blood pressure in nephritis and gout is due to the increased viscosity of the blood whereby it passes through the capillaries with greater difficulty and the heart action must be increased to overcome the increased peripheral resistance. The retention of lime in the aged increases the viscosity of the blood and this contributes to raise the blood pressure by increasing the peripheral resistance. In addition to this, there is usually a hypertrophied heart, apart from the atonicity of the vessels. A diminished amount of blood in the arteries has a counteracting influence but not enough to overcome the causes of increased blood pressure. When decompensation sets in the blood pressure falls.

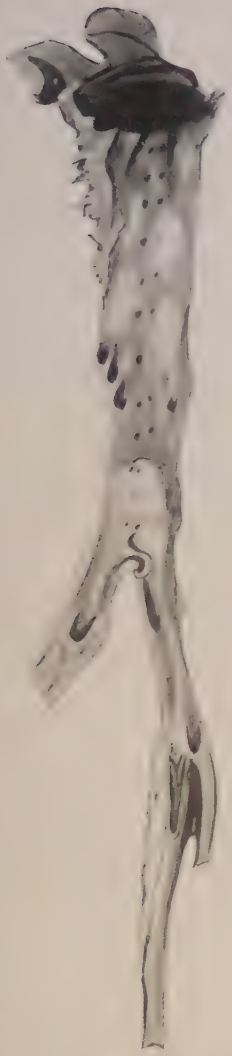
In some cases the earliest sign of arteriosclerosis is seen in the tortuous retinal vessels. Occasionally the normal difference in the pulse rate when standing and when in the recumbent position is not maintained. If the pulse is more frequent in the recumbent position the disease is well advanced. The disease may be suspected if the rate when standing is less than six over

the pulse rate when lying down. There are numerous vague symptoms of nervous origin which have been considered suggestive of approaching arteriosclerosis and their presence has given rise to the opinion that there exists a presclerotic stage of the disease. When we remember that the normal degeneration is slowly progressive, that there is a progressive increase in blood pressure from birth and that the normal condition is a disease condition only in the sense that it produces *discomforts* we will realize the impossibility of determining at what point senile arteriosclerosis becomes pathological, or of defining the so-called presclerotic stage. Such symptoms as headache after smoking, palpitation upon exertion, sensory disturbances, etc., are early manifestations of localized arteriosclerosis; they may, however, be due to other conditions than arterial degeneration. A fairly reliable premonitory sign of diffuse arterial degeneration is an intermittently high blood pressure, the pressure rising higher from ordinary causes producing temporary tension than the cause itself would warrant. In a typical case a man aged sixty had a normal systolic pressure of 160 mm. upon arising. Walking up one flight of stairs raised the pressure to 190 and half an hour afterward the pressure dropped to 150. There was no other sign of cardiac or vascular disease but six months later the man had a stroke of apoplexy.

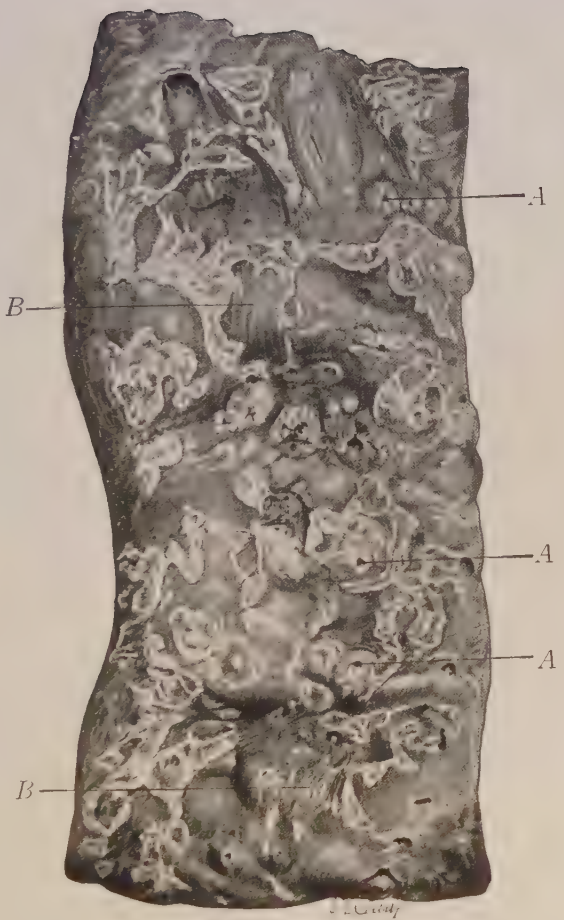
For the purpose of systematizing the manifold symptoms of regional arteriosclerosis the disease will be divided into central, including the heart and large vessels; visceral, including the viscera and the vessels supplying them; and peripheral arteriosclerosis, including arterioles and the peripheral vessels.

Senile endocarditis and the various cardiac lesions due to arteriosclerosis ought to be included in the central group but will be described separately.

Aortic Arteriosclerosis.—Aortic arteriosclerosis is the most frequent of the arterial degenerations. The vessel is dilated, its expansibility is diminished, the muscular coat is replaced by fibrous tissue and there are fatty or calcareous plaques on the inner coat. It gives no distinctive symptoms. Occasionally an increased area of dullness can be demonstrated, there is sometimes a systolic murmur over the vessel and there may be jugular pulsation. If the arch is involved the pulsation can sometimes be felt behind the suprasternal notch. There may be a dif-



Arteriosclerosis of Abdominal Aorta. (Satterwaite *Medical Record*, May 14, 1910.)



Aorta, Opened, Showing Different Types of Atheroma. (From Coplin's "Manual of Pathology.") The surface is most extensively altered by infiltration, degeneration, and necrosis. Many of the necrotic areas are calcified and could be fractured by bending. *A*, *A*, *A*. Elevated obstructing patches of atheroma surrounding exit points of small branches. *B*, *B*. Linear atheroma.

ference in the character of the carotid pulse on the two sides. A difference in the character of the radial pulse on the two sides may be due to aortic, subclavian, brachial or radial arteriosclerosis.

Acute Degenerative Aortitis.—This occurs most frequently in plethoric individuals showing symptoms of cardiac hypertrophy, palpitation and dyspnea. The symptoms come on suddenly with intense dyspnea resembling a severe attack of spasmodic asthma, a pain over the aorta, anginal in character, during the attack, and a constant pain or ache between the attacks. In some cases the first attack destroys life, more often the patient succumbs after several attacks.

Aortic Aneurysm.—Aortic aneurysm may appear as a manifestation of senile arteriosclerosis, although it occurs more frequently as a result of syphilitic infection. Occurring as a primary disease it is generally spindle shaped, the dilatation involving all the walls of the vessel. Its progress is slow when due to senile arteriosclerosis, the dilatation proceeding but little faster than the dilatation of the rest of the vessel, and the walls of the aneurysm continue to harden with the walls of the aorta above and below it. For this reason it never attains the size of the syphilitic aneurysm nor of the aneurysm due to traumatism or sudden strain, and the pressure symptoms that mark the other forms are seldom pronounced. The lesion is most frequently at or just above the root of the ascending portion of the aorta and is recognized more often by the physical signs than by pressure symptoms. There is usually a palpable occasionally a visible, pulsation over the site of the dilatation, dullness on percussion and a systolic murmur carried upward to the neck. When the arch is involved the signs are most pronounced behind and to the left of the upper part of the sternum, and when in the descending portion the signs are most pronounced in the interscapular space. Bretschneider reported a case of sclerosis of the arch of the aorta which presented as one of the symptoms, an intermittent dyskinesia with paroxysms of pain along the arm, numbness and loss of contractility of the muscles of the entire upper extremity.

Coronary Arteriosclerosis.—Sclerosis of the coronary arteries has no pathognomonic symptoms. The only symptom which is constantly associated with this condition is angina pectoris, but

this symptom may be due to other causes and coronary sclerosis has been found after death which gave no symptoms during life. As one result of coronary disease is malnutrition of the heart with consequent muscular degeneration, the symptoms of such degeneration whether fatty infiltration, fatty degeneration, myofibrosis or brown atrophy, point to coronary sclerosis. The symptoms associated with angina pectoris will be given in describing this disease under cardiac neuroses. The presumptive diagnosis based upon cardiac asthma, cardiac degeneration and angina pectoris amounts almost to a certainty.

Arteriosclerosis of the Pulmonary Artery.—Arteriosclerosis of the pulmonary artery is rare and the diagnosis is difficult. There is generally a history of infection with mitral stenosis or aortic insufficiency and arteriosclerosis of the aorta. The symptoms are cyanosis without dyspnea or edema, and pulmonary hemorrhage. The physical signs are, an area of dullness about the upper left margin of the sternum sensitive to pressure and percussion, cardiac dulness increased to the right, the diastolic thrill and the presystolic murmur of mitral stenosis above and to the right of its usual location. The condition may be diagnosed by radiography.

Arteriosclerosis of the Abdominal Aorta.—A positive diagnosis of sclerosis of the abdominal aorta can be made only when the abdominal walls are thin and the artery can be felt. It may be possible then to feel nodules or areas of hardness, or irregularities in the impulse given to the fingers by the pulsation of the vessel. There are no pathognomonic symptoms but the disease may be suspected when there are other symptoms and signs of a generalized arteriosclerosis and vague, painful sensations in the abdomen increasing at times to agonizing crises.

Cerebral Arteriosclerosis.—Cerebral arteriosclerosis gives somatic and mental symptoms. The earliest of the somatic symptoms is a dull frontal headache most severe upon arising and passing off in the course of the day. This headache is due to a passive hyperemia produced by the recumbent position, which passes away when the patient is erect. In some cases the cerebral hyperemia, caused by the recumbent position and relieved by the erect posture, is replaced toward evening by a gradually increasing cerebral anemia until a feeling of faintness compels the patient to lie down. Dizziness and vertigo are

early symptoms. There is a momentary feeling of fulness as though a gush of blood came up from below, went whirling through the head then just as suddenly dropped down again. This is accompanied by a flushing of the face, roaring in the ears, dimness of vision and dulness of intellect. The whole syndrome lasts but a moment and disappears completely. As the disease progresses the symptoms become more frequent and prolonged and during the attacks the patient becomes unsteady on his feet and may fall. Numbness, muscular twitching, weakness of the limbs, trembling and disturbances in articulation may occur. Insomnia is a frequent accompaniment. The mental disturbances are alternating depression and excitability, illusion and dementia. The illusions are not insane illusions but perverted perceptions which the patient recognizes as such. Thus asclerosis of the retinal vessels more marked on one side may give two different visual impressions, and an object may appear double, distorted or with a halo or shadow about it. There may be similar auditory perversion producing confused sounds. The patient knows that these are illusions and ascribes them to disease of the eye or ear. More serious are the mental confusion and delusions which occur when the patient becomes excited as these may persist and give rise to anxiety and other psychoses followed by melancholia and dementia.

In the severer forms of cerebral arteriosclerosis there may be transient paralysis, aphasia, hemianopsia, mental aberration, etc., passing away in a few hours. There is always danger from rupture of a minute vessel, producing apoplexy or from thrombus or embolus with consequent rapid cerebral degeneration.

Ear Symptoms.—The ear symptoms begin in a unilateral, later bilateral, tinnitus, followed by slight and progressive deafness, loss of air and bone conduction, dizziness and auditory illusions and hallucinations.

Eye Symptoms.—The arteriosclerotic eye shows tortuous retinal vessels, and occasionally retinal hemorrhages due to increased tension in vessels in which there is an endarteritis. There may be an embolus or thrombus of the central artery if there is cerebral arteriosclerosis and then the vessels are anemic. Usually there is a retinitis if the arteriosclerosis is associated with albuminuria, embolism or thrombus if associated with

cerebral or cardiac disease and atrophic choroiditis if associated with disease of the liver.

Abdominal Arteriosclerosis.—Degeneration of the abdominal vessels may be suspected when there are signs of diffuse arteriosclerosis and symptoms of visceral disturbances, non-inflammatory in character with progressive impairment of function. The symptoms of abdominal arteriosclerosis are more marked in allied organs and tissues than in the rest of the body and as the heart is usually the first to be affected by impaired circulation, the kidneys are soon involved. The kidney is frequently found degenerated yet the renal artery is not affected. (This will be taken up under senile degeneration of the kidney.)

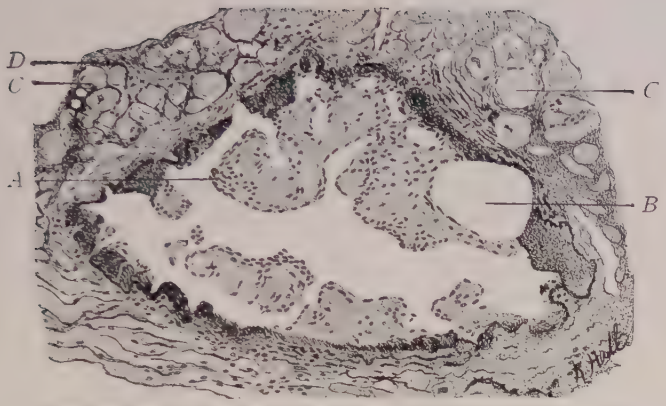
Gastro-intestinal Arteriosclerosis.—The symptoms of gastro-intestinal arteriosclerosis are manifold and appear to be due to impaired nutrition of the organs and irritation of the abdominal sympathetic nerves. We thus get two sets of symptoms, functional impairment and nerve irritation, the latter probably caused by some toxic substance in the blood.

The early diagnosis of this condition is difficult, as the organs affected appear to be the original seat of disease until other symptoms and signs of arteriosclerosis are found. There is usually abdominal pain about the umbilicus, at first paroxysmal, later continuous. The digestion is slowed and there is a feeling of oppression in the stomach for several hours after eating. This is due to dilatation and atrophic catarrh of the stomach.

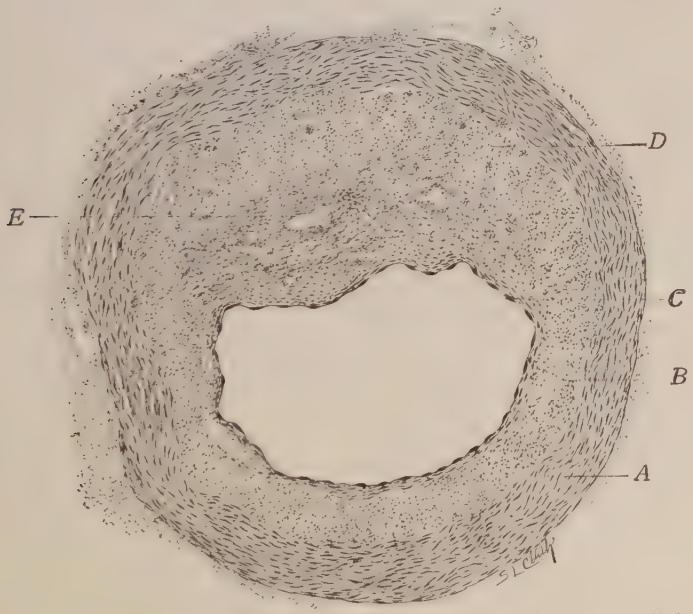
The intestinal symptoms are constipation, flatulence, meteorism, occasional watery or bloody diarrhea, beginning without apparent cause, lasting for a few hours there followed by constipation, sharp pains in the right hypochondrium, neuralgic but not colicky, occurring spasmodically and frequently at night. The mesenteric arteries are occasionally atheromatous but they give no pathognomonic symptoms. Lagane describes an arteriosclerotic syndrome of the intestines but the symptoms include many that are clearly due to other pathological conditions.

Some of the symptoms of abdominal arteriosclerosis resemble symptoms of tabes, colic, appendicitis, lead poisoning, nervous dyspepsia, neurasthenia, or other neuroses. In nearly all doubtful cases the etiological factors will clear up the diagnosis.

Hepatic arteriosclerosis.—While the liver generally shows atrophic degeneration due to malnutrition it gives no signs of this



Arteriosclerotic Disease of the Coronary Artery Giving Rise to Progressive Obliteration of its Lumen. (From Coplin's "Manual of Pathology.") Section taken from sclerotic periventricular branch shown in Fig. 230. The elastic lamellæ are fragmented, the endothelium has proliferated, and a forming thrombus is rapidly occluding the vessel. *A*. Forming thrombus covered at most points by endothelium. *B*. Channel through thrombus with partial wasting of adjacent vessel wall. *C, C*. Transverse section of muscle-fibers, showing fragmentation and retraction from the myocardial skeleton. *D*. Unusually conspicuous, apparently swollen elastica; the same change can be seen in many parts of the field. The fine stipple effect in the lower part of the figure, and especially marked in the lower right, is due to transverse sectioning of elastic fibers.



Coronary Artery, Showing Arterial Sclerosis. (From Coplin's "Manual of Pathology.") *A*. Adventitia. *B*. Media. *C*. Intima. *D*. Degenerating newly formed tissue which at *E* shows advanced softening.

condition except in diminished bile supply, the bile containing more cholesterin and sometimes producing the symptoms of cholecystitis and cholelithiasis. These will be taken up separately.

The pancreas and spleen, on autopsy, are often found degenerated, yet give no clear symptoms during life.

Ortner's Syndrome.—Ortner describes a symptom complex referable to the stomach, intestines, heart and lungs. These are distress after eating, distention of isolated section of the bowels with intense spasmodic pain, cyanosis and dypnea. He calls the disease " Dyspragia intermittens angiosclerotica intestinalis."

Arteriosclerosis of Spinal Vessels.—The dominant symptoms of arteriosclerosis of the spinal vessels are those of chronic myelitis, occasionally with symptoms of multiple sclerosis, syringomyelia, tabes dorsalis, or general paresis, without mental impairment. In rare cases there is a compression myelitis. Paralysis agitans, Charcot's claudication and senile tremor are supposed to be due to spinal degeneration following arteriosclerosis of the vertebral vessels, but these conditions are sometimes found in cases in which no spinal or arterial degeneration could be discovered. In *Charcot's claudication* there is an intermittent lameness following prolonged walking. The limb feels cold, there is rapid numbness, pain and sudden inability to move the limb. This passes off after rest but will return upon exercise of the limb. In this form of claudication the step is normal, but in other forms the step may be unsteady, short and tripping or slow, cautious, and long, dragging or jerking.

Peripheral Arteriosclerosis.—In peripheral arteriosclerosis the anatomical and functional changes occur in the muscles and skin. There is a sallowness resembling the cancer cachexia, local symptoms of numbness, coldness, tingling and other paresthesias, cramps, myalgia, pruritus, purpuric eruptions and other forms of skin disease. Senile gangrene is often due to localized arteriosclerosis and *Raynaud's disease*, when occurring in the aged, is supposed to be due to peripheral arteriosclerosis and neuritis. Many cases of senile gangrene give a history of local syncope and asphyxia preceding the gangrene and are really cases of Raynaud's disease in which the earlier symptoms were neglected.

Diagnosis.—The protean character of arteriosclerosis and the impossibility of differentiating between similar visceral symptoms

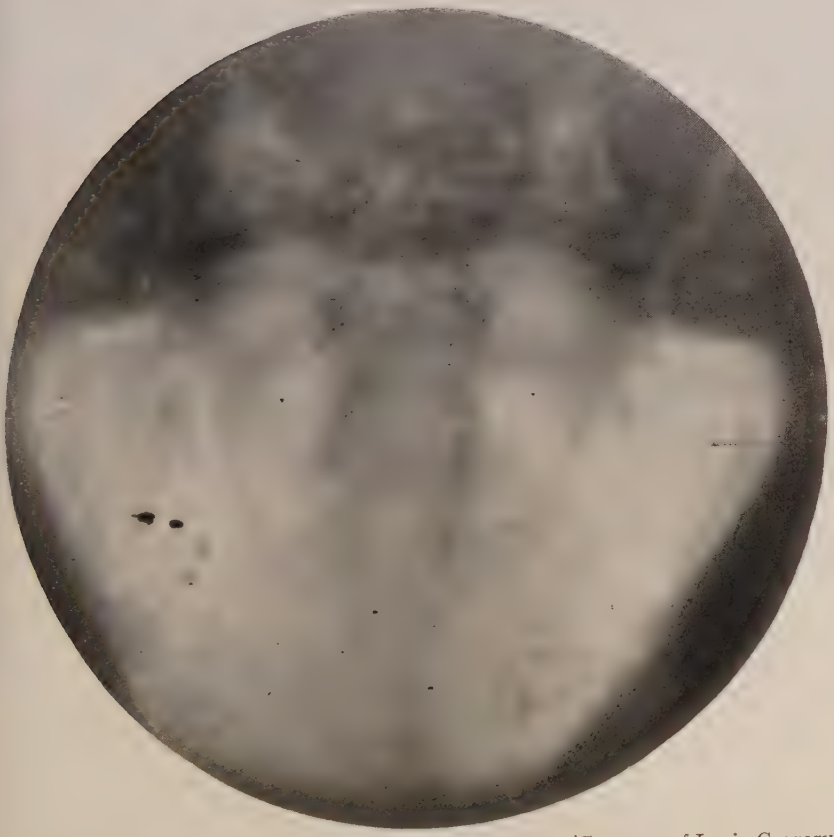
due to this and to other causes make diagnosis difficult and errors frequent. After the hardening of the radial artery has become so pronounced as to be recognized by the finger, the diagnosis is evident, but by this time the disease has advanced so far that nothing, or but very little can be done to improve the condition. An early diagnosis is of the greatest importance and in the absence of pathognomonic symptoms we must consider etiology, pathology, and general symptoms and we may be obliged to depend upon the result of treatment to prove the correctness of our diagnosis.

In senile arteriosclerosis the principal etiological factor is age. Then come occupation and mode of life, mental labor favoring cerebral arteriosclerosis, exciting or difficult physical labor predisposing to central arteriosclerosis, etc.

Early physical signs are cardiac hypertrophy, accentuation of the second sound of the heart and high blood pressure. In precocious senility, we have an unfailing sign in early ossification of the costal and xiphoid cartilages. The earliest symptoms will depend upon the location of the disease. If in the head, headache upon arising and occasional vertigo will be noticed. In the arms or legs there may be muscle cramps after exercise. In making our diagnosis of arteriosclerosis of an abdominal viscus we must be guided by the symptoms and signs of generalized arteriosclerosis. In the absence of such symptoms and signs if the ordinary treatment for the local conditions is ineffectual a single dose of nitrite of soda or nitroglycerin may clear up the diagnosis by giving immediate relief. This is of especial service in the diagnosis of cases involving spinal symptoms. Many of the symptoms given above may be due to antecedent disease to which the arteriosclerosis is secondary and it is not unusual to have the cause for secondary arteriosclerosis prevail in old age. It is thus possible to have a primary senile degeneration and a secondary degeneration of the arteries following syphilis, gout, and infectious disease, etc., prevail at the same time, either affecting different organs or one aggravating the other.

Prognosis.—The prognosis of senile arteriosclerosis is unfavorable. It is progressive and generally destroys the individual either through exhaustion of the heart or through impairment of some other organ to such extent as to prevent its functions or through cerebral compression following cerebral

Atheroma of Brachial Artery. (Pic and Bonnamour.)



Radiogram of Arteriosclerosis of Internal Iliac Artery. (Courtesy of Louis Gregory Cole, M. D. New York.)

hemorrhage. Occasionally an embolus blocks a vessel and prevents the nutrition of a part causing its rapid degeneration, or in the case of the lungs causing fatal dyspnea. In the case of embolism, death may also be due to shock. General exhaustion due to profound changes in the organs and nerves controlling metabolism can generally be traced to arteriosclerosis.

The progress is normally slow but is hastened by improper living, poor quality of food, lack of exercise and vitiated air. Cardiac hypertrophy keeps pace with the impairment of the circulation due to degeneration of the vessels. With the limit of hypertrophy is also reached the limit of tonicity. Further strain results in broken compensation and dilatation and this ends in cardiac exhaustion.

In the secondary arteriosclerosis, the prognosis will depend upon the cure of the primary disease and the activity of the metabolic processes. In young individuals in whom the anabolic processes surpass the destructive forces, the inhibition of the cause and elimination of waste will prevent further degeneration and remove the pathological tissue which will be replaced by new tissue. In senile cases the metabolic processes proceed slowly, and as the repair is accomplished through the blood which is deficient in quality and quantity, a lower type of tissue is formed to replace the degenerate tissue. In this way the character of the tissue is altered through the proliferation of connective tissue and waste of normal substance.

Treatment.—Senile arteriosclerosis being a natural, normal condition is incurable in the sense that it can neither be prevented nor removed. The best that we can hope for is to retard its progress and relieve disagreeable symptoms.

Before beginning treatment we must be certain that the condition is not a secondary disease. If the patient has ever had syphilis, acute articular rheumatism or gout, though the symptoms had disappeared years before, drug treatment applicable to the primary disease should be instituted. In these cases the iodides are serviceable. It does not matter in what form the iodine is introduced whether in the form of inorganic salts or organic preparations the effect upon the blood-vessels and the blood is the same. The objection to the iodide of potassium is its irritating effect upon the gastric mucous membrane. This is not as pronounced if the iodide of sodium is

used. The iodide of arsenic in 1/15-grain doses is the most valuable of all the inorganic iodine compounds, the arsenic being a tonic and an anabolic stimulant. It is, however, eliminated slowly and produces cumulative toxic effects. If it is employed, it must be discontinued as soon as the physiological effects of arsenic—gastric irritation, metallic taste or swelling under the eyelids—appear. Some of the organic iodine preparations do not affect the stomach, nor do they produce rashes, nasal or bronchial catarrh or other untoward effects. The extravagant claims made by the manufacturers of these organic compounds deter the author from recommending any one in particular. The iodides should never be used if there is high blood pressure and low viscosity of the blood as the iodides still further lower the viscosity thereby favoring hemorrhage, especially miliary hemorrhages from the cerebral vessels. Lime salts are positively contraindicated there being already an excessive retention and diminished elimination of calcium. In senile arteriosclerosis no drug will permanently improve the condition of the arteries. Whatever permanent benefit can be brought about must come from the regulation of the mode of life of the individual. Drugs must however be employed to relieve the disagreeable concomitants of arteriosclerosis either by lowering the blood pressure or by local treatment of the part giving the disagreeable symptoms.

Many drugs will lower blood pressure yet some in doing so produce direct or secondary effects more serious than the condition they are intended to relieve. Aconite, gelsemium and veratrum viride lower blood pressure by depressing the heart, weakening its force and slowing its action thereby diminishing the blood supply to organs and tissues already impaired through insufficient nutrition. These drugs should be used only when there is a rapid and full pulse not due to nervous causes and then they should be combined with a cardiac stimulant which has no vasoconstrictor effect.

The choice of drug to diminish the blood pressure must depend upon its action upon the heart, the vessels and the blood.

The favorable action of the iodides is due to the property of reducing the viscosity of the blood, thereby allowing the blood to flow more freely through the contracted vessels. It has been suggested that they stimulate metabolic activity either by direct

action upon degenerate tissue or by stimulation of the thyroid gland. They are useless when the degeneration is part of the normal process of involution and even small doses will produce the physiological effects, iodism with rashes, catarrhs and local irritation. The calcium compounds of iodine have the additional disadvantage of increasing the viscosity of the blood and furnishing an excess of calcium.

The most valuable drugs to reduce blood pressure are the nitrites which act by dilating the arterioles. In angina pectoris where rapid action is required amyl nitrite used by inhalation produces an almost instantaneous effect, lasting, however, but a few minutes. Nitroglycerin in doses of one or two minims of a 1 per cent. solution acts almost as rapidly when given hypodermically and somewhat slower when given by mouth. The action lasts about fifteen minutes. The nitrite of soda in grain doses acts in fifteen to twenty minutes and its action lasts two or three hours. Erythrol tetranitrate has been recommended on account of its more prolonged action, but it possesses no other advantage. It is given in $\frac{1}{2}$-grain doses. The dose of the nitrites depends to a great extent upon the tolerance or idiosyncrasy of the patient and the condition of the cerebral vessels. If there is cerebral hyperemia a sudden influx of blood may cause rupture of a vessel. The long-continued use of the nitrites may cause peripheral stasis while the drugs themselves act as blood toxins. Thyroid extract controls high blood pressure and has been highly extolled in the treatment of arteriosclerosis. In the author's experience small doses produced distressing palpitation of the heart and the blood pressure rose within a day after its use was discontinued.

Theobromin and its combinations have been employed to reduce blood pressure. Huchard recommends its use as it dilates the peripheral vessels and stimulates the heart and the kidneys. Diuretics lower blood pressure through the abstraction of fluid from the circulation. This, however, is a disadvantage since it increases the viscosity of the blood and the system demands restitution in thirst which cannot be appeased until sufficient fluid has been imbibed to supply the deficiency.

Electrotherapeutists report reduction of blood pressure through high-voltage currents, but this method of treatment is

experimental and there are divergent views as to the utility of electricity in this condition.

Among general measures employed in arteriosclerosis, Trunecek's serum deserves attention. This is a solution of the various salts in the proportion in which they are found in the plasma. The theory of its employment is based upon Weil's theory of disproportion of salts in the blood, Trunecek arguing that by administering the plasma salts the plasma would finally hold the normal proportion, any excess of one or more salts being eliminated by the kidneys. It is believed that decalcification of the vessels cannot be accomplished by chemical means, and consequently such result, if accomplished at all, must be brought about through altered metabolism. This is the only explanation that can be given for the favorable results obtained in many arteriosclerotic cases treated by the Trunecek serum or salts. (The salts are marketed under a trade name.)

In treating local conditions the effect of drugs rarely lasts longer than the period of effectiveness of the last dose. If there is pain morphine will relieve it but if the cause persists the pain will return. For insomnia 5 to 10 grains of veronal may be given at bed time but care should be taken that the bladder is emptied. A hot foot bath in the morning will relieve the morning headache and a whiff of ammonia or thirty minims of the aromatic spirits of ammonia can be used for the vertigo. The attacks of vertigo usually last but a moment and pass away before treatment can be instituted. Muscle cramps in the limbs will disappear if hot water or hot cloths are applied and the same treatment will generally relieve claudication. (Treatment of other local conditions will be given under the description of such conditions.) Hygienic and dietetic measures take first place in the treatment of arteriosclerosis as the disregard of such measures is mainly responsible for this disease or its early appearance.

The most important rule is the diminution of food to the amount actually required to maintain strength, the elimination as far as possible of purin-forming protein foods, a minimum of lime-containing foods and of those containing much cellulose and other indigestible material. The amounts should not be left to the judgment of the individual but it should be regulated as

Arteriosclrosis of Posterior Tibial Artery. (Courtesy of Lewis Gregory Cole, M. D., New York.)

carefully as in the treatment of diabetes. The amount of tea and coffee should be cut down but they need not be entirely eliminated. Alcohol is injurious yet the sudden and total deprivation of alcohol to a person accustomed to it will produce mental depression. It should be cut down gradually. Beer is worse than spirituous liquors while light dry wines are the least objectionable.

The loss of the teeth in old age is a physiological indication that foods requiring thorough mastication are unsuitable and this applies especially to meat. If there is evidence of intestinal decomposition in foul-smelling stools, intestinal antiseptics are required. For temporary use we can employ salol, salicylic acid or the sulpho-carbolates. For prolonged use the lactic acid ferments are best. When the disease is far advanced and there is broken compensation and renal difficulties milk must form the principal article of diet. Non-alcoholic malt extract is a valuable adjunct to the food of the aged.

It is hardly necessary to insist upon a daily evacuation of the bowels.

Many writers dilate upon the injurious effects of smoking yet most old men are smokers. Excessive smoking as well as excesses in other things is injurious but it need not be forbidden entirely unless headache or vertigo follows.

Excessive physical exercise should be forbidden and it should be an imperative rule for the patient to rest as soon as he begins to feel fatigued. Further exertion can be carried on only under a forced impulse which strains the heart, increases the circulation abnormally and hastens degeneration of the vessels and organs involved. A sudden exertion or intense excitement is liable to cause paralysis of the heart. Mild mental and physical labor is beneficial before myocardial incapacity has set in, but afterward complete mental and physical rest is necessary. In the early stage active elimination of toxic material by catharsis, diuresis and diaphoresis is advisable. Later on the eliminative treatment should be continued but not forced unless local conditions such as constipation or enuresis make it necessary. Extreme changes in temperature, in climate, in air pressure as when going from the seashore to the mountains, should be avoided. A cheerful spirit, keeping the mind pleasantly employed and free from worry, and the kindly ministration of

the family and the physician often do more to relieve the disagreeable symptoms than drugs or other measures.

Amorphous Phosphorus in Senile Arteriosclerosis.—The author has used the red amorphous phosphorus in senile arteriosclerosis for several years. Given originally as a substitute for ordinary phosphorus in senile debility it was found that it was eliminated as amorphous phosphate of lime and that the lime elimination was thereby increased. Weil's experiments showed that the lime elimination in arteriosclerosis was diminished. Phosphorus has the property of combining with lime and increasing the lime assimilation. In the small doses which can be given when the ordinary phosphorus is employed the phosphorus will combine with the lime of the food and increase the amount of lime salts in the body. When given as amorphous phosphorus the dose is two grains or more several times a day, and with a lime-free diet the lime required for the combination necessary to secure the elimination of the phosphorus excess, is drawn from the abnormal lime deposits. This appears to be the rationale of the treatment and explains the good results obtained from its use.

SENILE PHLEBOSCLEROSIS

Phlebosclerosis is a degeneration of the walls of the veins analogous to the degeneration of the arterial walls.

Etiology.—A primary senile degeneration of the veins is rare and occurs almost exclusively in veins subjected to great pressure such as the veins of the lower extremities. The disease generally follows a phlebitis, a pyemia or an infectious disease and may then occur at the site of the phlebitis, or elsewhere. If there is no antecedent disease, the degeneration is due to impaired nutrition through contracted vasa vasorum as occurs in arteriosclerosis.

Pathology.—Primary degeneration through impaired nutrition presents similar histological changes as are found in arteriosclerosis. The elastic and muscular fibers waste and permit dilatation of the vesesl and the production of varicose veins. This causes slowing of the current with production of thrombi in the pouches of the varicosed portions of the vessels.

Symptoms.—Phlebosclerosis presents no marked symptoms. Sometimes areas of hardness can be felt along the course of superficial vessels, ordinarily, however, the symptoms are those of varix. If lumps are felt in the varix dilations they are usually due to thrombi. A thrombus in a vessel which is not dilated, interferes with the circulation and causes local edema, becoming worse and accompanied by pain upon prolonged standing. A hypostatic edema of the ankles and feet, frequently met with in the aged, is probably due to phlebosclerosis. This edema is slight except after prolonged standing or walking. The physical fatigue produced by such exercise necessitates rest and thus any great accumulation of plasma in the subcutaneous tissue, and the discomfort which would accompany extensive exudation, is avoided.

In making a differential diagnosis of phlebosclerosis from this form of edema we must eliminate cardiac and renal disease, anemia, flatfoot and obesity. It may occur in varicose veins.

Treatment.—There is no treatment for phlebosclerosis. The treatment for venous thrombus and varicose veins is given under varicose veins. For the edema rest and rubber ankle supporters will give relief, but the condition progresses with the cause.

SENILE DEGENERATION OF THE HEART

The earliest cardiac change due to age is cardiac hypertrophy. This is not a senile change but the ordinary trophic increase in muscular development that occurs normally in all striped muscles actively employed. The hypertrophy caused by the greater effort of the heart to send the blood through vessels having diminished contractile power or diminished caliber, does not differ from the hypertrophy due to excessive exercise in earlier life. The athlete's hypertrophied heart remains enlarged for years after the athletic work has been given up and may become a permanent condition. The hypertrophy of the senile heart is confined to the left ventricle until the valves are involved.

There is a limit to muscular capacity and when this has been reached further activity will cause muscle exhaustion or degeneration. The muscle can recover from exhaustion upon com-

plete rest but as the heart cannot absolutely rest, degeneration takes place. The usual degenerative change in these cases is a loss of tonicity of the muscle fiber whereby its irritability and contractility are diminished and it stretches, permitting a dilatation of the cavity. While dilatation of the heart is the most frequent sequel of cardiac disorders peculiar to the aged, it is not a senile degeneration and will therefore be discussed in the fourth group. The senile myocardial degenerations are myofibrosis and brown atrophy, the former occurring most frequently when the hypertrophied heart has been reached the limit of its functional capacity and further strain causes exhaustion, atonicity and degeneration. Brown atrophy occurs most frequently in hearts that have not been greatly hypertrophied, but in which coronary arteriosclerosis appeared early and interfered with the nutrition of the organ. Myofibrosis is therefore a mechanical degeneration, while brown atrophy is a nutritional degeneration. Other degenerations, though frequent in the aged, are not strictly senile processes.

Senile Myofibrosis

Senile myofibrosis, erroneously called chronic myocarditis, is a degeneration of the cardiac muscle marked by an increase of the interstitial connective tissue with waste of muscle fiber. It corresponds to the nutritional type of arteriosclerosis. It is the usual form of senile degeneration and in its milder form is normal.

Etiology.—Myofibrosis is due to impaired nutrition either from some fault in the blood or from diminished supply through coronary sclerosis.

As myofibrosis is one of the terminal results of malnutrition whether due to impaired quality of the blood or to diminished quantity, anything which will cause either of these may produce a myocarditis and consequent degeneration. The diminished blood supply causes insufficient repair of muscle waste, but there is sufficient to supply hyperplastic connective tissue which requires less nutrition or perhaps the blood of the aged contains more of the nutritional elements required by the connective tissue and less of the elements required to repair muscle waste. This would explain the general tendency to fibrosis in old age.

Satterthwaite has shown how an embolus from chronic endocarditis might be arrested in a branch of the coronary artery and produce local infarct with fibrosis. Chronic endocarditis may follow an acute myocarditis, endocarditis, pericarditis, infectious disease, syphilis, gout, nephritis, diabetes, or alcohol, lead, or tobacco intoxication.

Pathology.—Senile myofibrosis affects the whole organ but the hyperplasia is most marked in the auricles. In the early stage of the disease the heart is usually enlarged, hypertrophied or dilated, later as the muscle waste proceeds it becomes smaller. There is an increase of connective tissue and a waste of muscle fiber, but the muscle fibers are *not* infiltrated with granular matter as occurs in myocarditis following infectious diseases and toxins. They may present segmentation and fragmentation. The heart feels harder and when cut across it looks lighter in color than normal. The valves are usually affected but this may be due to a senile endocarditis or to other senile changes. In some cases there is in addition to the fibrosis a fatty degeneration of some fibers. It is not known what particular factor determines the character of the degeneration, fatty or fibrous, when the blood supply is diminished. As there is in both the same underlying etiological factor—insufficient blood supply—the determining factor must be sought for in the blood itself. Dilatation is a frequent sequel.

Symptoms.—Senile myofibrosis produces progressive heart weakness. The force of the contractions is diminished and slight causes will produce arrhythmia and palpitation, while intense excitement may produce delirium cordis, spasm or heart block. In the mild form there may be no symptoms except perhaps palpitation, vague precordial distress and dyspnea upon slight exertion, but these symptoms may be so mild as to pass unnoticed. When the disease is further advanced there is usually a weak irregular pulse and weak apex beat, the symptoms of imperfect aeration, dyspnea and cyanosis with headache and the symptoms of cerebral anemia, facial pallor, blanched conjunctivæ, and a feeling of emptiness in the back of the head with occasional vertigo, and the symptoms of surface anemia, pale, cold, dry skin. Irritability of temper is frequent. Other organs become affected through impaired circulation, insufficient blood supply and passive congestion. Neurasthenia occurs

frequently and angina pectoris occasionally, due to coronary sclerosis.

Diagnosis.—While the various forms of cardiac degeneration present differences in their pathology, it is often impossible to distinguish between them clinically. In many cases the history will determine the diagnosis. Any cause which will produce an acute myocarditis will produce a chronic myocarditis with consequent fibrosis. The acute myocarditis is, however, very rare in the aged and is almost always a secondary infection or intoxication most frequently following an influenza. The chronic myocarditis is secondary to acute myocarditis from toxin or gout, rheumatism, etc. In senile myofibrosis the antecedent relations are cardiac hypertrophy and coronary arteriosclerosis, the former causing degeneration through overactivity and consequent exhaustion, the latter through malnutrition. The symptoms of acute myocarditis are pain and a feeling of oppression over the heart, anxiety and a fear of death, feeble and irregular heart action, the pulse small, irregular and gradually weakening. In chronic myocarditis the symptoms of the acute form appear milder and more persistent, there is dyspnea and later cyanosis. It is often difficult to differentiate between chronic myocarditis and senile myofibrosis except by the history and by the course of the disease. Senile myofibrosis is progressive and while its progress may be retarded it cannot be halted while myocarditis may be cured. In both diseases the symptoms improve after prolonged rest but in senile fibrosis the heart becomes weak again after exercise.

Fatty degeneration produces similar symptoms but the heart is persistently weak while in fibrosis the heart is sometimes fairly strong, especially after prolonged rest. In brown atrophy the size of the heart is diminished and symptoms of coronary arteriosclerosis are frequent while in myofibrosis there is usually enlargement of the heart and coronary symptoms are infrequent.

Treatment.—The treatment of senile myofibrosis must be hygienic and symptomatic. The condition is slowly progressive and we can do nothing to arrest its progress nor to restore degenerate tissue. The symptoms are usually so mild that they are neglected by the patient and are but mentioned incidentally when complaining of the more distressing symptoms of some other disease. (The treatment of the incidental symp-

Arteriosclerosis of Peroneal Artery. (Courtesy of Lewis Gregory Cole, M. D., New York.)

toms arrhythmia, palpitation and angina pectoris will be given under Cardiac Neuroses.)

When cardiac weakness becomes marked the most important measure is rest. Digitalis is contraindicated as the degenerated fibers cannot respond and the excessive work imposed upon the healthy fibers causes their rapid degeneration while its vasoconstrictor action further diminishes the blood supply to the heart by contracting the coronaries. Where there are marked symptoms of cerebral and peripheral anemia the nitrite of soda in 1/6-grain to 1-grain doses four times a day should be given. Alcohol in the form of whiskey or wine given with meals is often beneficial. Late in the disease camphor, caffein, strychnin and strophanthin may become necessary. In regulating the life of the patient the avoidance of sudden strain is of the greatest importance. Even straining at stool is injurious and may cause sudden cardiac exhaustion. A profound emotion may do the same. Some exercise is necessary but it must not produce fatigue or strain and it must be stopped as soon as dyspnea or palpitation appear. The rarified air of the highlands is detrimental. The Schott, Nauheim and Oertel treatment are dangerous and should not be used in senile cases notwithstanding favorable reports from those interested in institutions giving such treatments.

Dietary restrictions are directed principally to non-constipating foods. Sexual excitement should be avoided. (The treatment for coronary arteriosclerosis is given under Arteriosclerosis.)

Brown Atrophy of the Heart

Brown atrophy is an infrequent physiological, atrophic condition of the senile heart. It must be observed that degeneration of the heart muscle occurs normally much later than the degeneration in other organs and tissues and upon autopsy of aged individuals we frequently find no cardiac change other than hypertrophy or dilatation with the accompanying valvular lesions and perhaps a senile endocarditis.

Etiology.—It occurs normally if there is a slowly developing coronary arteriosclerosis without marked hypertrophy. It has been found also in younger individuals who were suffering from prolonged toxemias, tachycardia and overwork.

Pathology.—In brown atrophy the heart is diminished in size and there is little or no hyperplasia of connective tissue. The muscle cells are atrophied and there is a deposit of brownish pigment about the nuclei. The muscle striations become obscure but segmentation and fragmentation does not occur. The heart is of a dark brown color and may appear shrunken and withered with the vessels brought out in relief.

Symptoms.—The symptoms are those of cardiac weakness. There is a weak apex beat and weak pulse becoming irregular in rhythm and force upon exertion or excitement. At such times there are also dyspnea and palpitation and the patient becomes irritable. In advanced cases there are the symptoms of imperfect aeration, cerebral and peripheral anemia, and functional impairment of other organs and tissues through impaired circulation and passive congestion. The symptoms are the same as in other forms of myocardial degeneration and the diagnosis will depend upon the diminished area of cardiac dulness, the age and the exclusion of other causes of cardiac atrophy, starvation and wasting diseases. The atrophic stage of senile fibrosis occurs late, perhaps years after the earlier symptoms called attention to this condition.

Treatment.—What has been said of treatment under senile fibrosis applies to brown atrophy. Hygienic measures, especially the avoidance of physical exertion, strong emotions and excitement are more important than drugs.

SENILE ENDOCARDITIS

This form of chronic endocarditis is a senile degeneration of the endocardium and is usually part of a general arteriosclerosis. It cannot be differentiated except etiologically from the chronic endocarditis that follows the acute form and from the sclerotic endocarditis that is induced by alcohol, syphilis, autogenous toxins, excessive exercise, etc.

Etiology.—When part of a general arteriosclerosis, there are the same etiological factors (see senile arteriosclerosis). In rare cases there is an extension of the sclerotic process from the aorta to the aortic valve and to the left ventricle.

Pathology.—The changes in the endocardium are like the degenerative changes in the endothelial layer of the blood-vessels.

The membrane becomes thickened and firmer, there is frequently a covering layer of fibrin over atheromatous patches and occasionally calcareous plates are found. The changes are most pronounced about the valves which become thickened and misshapen and lose their elasticity. The cordæ-tendinæ become sclerosed. The aortic valve is most frequently involved, the structural changes producing insufficiency.

Symptoms.—This condition cannot be diagnosed until the valves are involved, when the symptoms of valvular disease appear. There may be occasional precordial distress, arrhythmia, palpitation, etc., but nothing distinctive upon which to base a diagnosis.

Treatment.—The treatment depends upon the valvular disease that is produced.

SENILE DEGENERATION OF THE LUNGS

Senile atrophy of the lung is a physiological condition, but it may produce so much distress as to require medical intervention.

Etiology.—A number of causes combine to bring about pulmonary degeneration. Apart from theoretical causes, tissue-cell evolution and a change in the character of the blood, arteriosclerosis of the pulmonary vessels diminishes the quantity of blood sent through the lungs. Pneumokoniosis stimulates connective-tissue hyperplasia and the contraction of this fibrous tissue compresses the lung tissue. The lungs are further compressed by the rigidity of the chest walls. There is then insufficient nutrition and mechanical compression, causing waste of tissue.

Pathology.—The lung undergoing senile degeneration is smaller than in maturity, discolored, and on section presents minute cavities. These are due to the waste of the alveolar septa whereby the air cells coalesce and the condition called *small chest* or *senile emphysema* is produced. The residual air is increased, the vital capacity is diminished and both inspiration and expiration are lessened.

Symptoms.—The symptoms of senile degeneration of the lungs become distressing when there is marked emphysema.

The diminished respiration and lessened aerating surface cause incomplete aeration of blood and consequent cyanosis but this produces very little or no distress. The real distressing symptom of pulmonary degeneration, "dyspnea," is due to senile emphysema.

Senile emphysema differs from the emphysema of maturity in the absence of the barrel-shaped chest which is almost pathognomonic of this disease in earlier life. In the senile form the chest walls are rigid, there is no respiratory expansion and the respiration is carried on mainly by the diaphragm. When the dyspnea is severe, there is a raising and dropping of the entire thorax, the supra- and infraclavicular spaces sink and the muscles of the neck are prominent. Percussion shows that the lungs lie lower than in maturity, the apex is lower and the percussion here is indistinct. The percussion note in senile emphysema is peculiar, there being a typanitic resonance without the momentary echo sound that accompanies the hyperresonance of ordinary emphysema. Upon auscultation we get a weak vesicular murmur with prolonged expiratory note. If the patient has been lying upon his back, immediately upon arising dry crepitant râles can be heard at the lower part of the back during the first few inspirations. This is due to the opening of the air vesicles in that part of the lungs, which were compressed in the recumbent position and is a pathognomonic sign of this condition.

Moist râles are due to bronchitis. The latter if present is not associated with senile emphysema as in other forms of emphysema, but is an accidental complication. The dyspnea of senile emphysema is both inspiratory and expiratory, the latter more pronounced. In the early stage it appears only upon excessive exertion, later slight exertion as walking up a few steps, may bring it on. When this occurs there is extensive involvement of the lungs, with cyanosis and cardiac disease. Usually senile emphysema gives no symptoms until the disease is far advanced, as the physical condition of the individual prevents him from undertaking difficult tasks which might cause dyspnea.

Treatment.—Prophylactic measures can be employed to defer the atrophy of the lungs. A cane should be used as soon as it is noticed that the ageing individual walks with a stoop. Shoulder braces are useful but irksome. Deep breathing, taking a long, steady deep breath should be practiced several times a day.

When seated, the person should use an arm chair and keep his arms upon the rests. A warm, dry, equable climate at low elevation and free from dust is the most important hygienic measure. Mild exercise is beneficial but fatigue must be avoided. The individual should change his position frequently, alternating between walking, standing, sitting and lying down. Belladonna is useful as a stimulant to the respiratory centers and oxygen inhalation for cyanosis.

PNEUMOKONIOSIS

Pneumokoniosis or fibrous induration of the lung is caused by the constant inhalation of dust.

Etiology.—Dust is inhaled in the air stream from birth. Most of the dust is caught by the ciliated epithelium of the bronchi, but some reaches the alveoli and works its way or is possibly carried by phagocytes into the lymph spaces and connective tissue, causing a chronic irritation with consequent hyperplasia of such tissue. Some induration from this cause is found in every aged individual. It is more pronounced among city dwellers, especially in manufacturing towns in which there is much soot and dust in the atmosphere, and occurs to but a slight extent in sailors of sailing vessels.

Pathology.—The lungs are discolored, the pigment ranging from gray to black, depending upon the character and quantity of dust that had been inhaled. This discoloration may be uniform throughout the lungs or appear in scattered areas giving the lung a mottled appearance. There is a hyperplasia of connective tissue, the new tissue being pigmented. Dust particles may be found in the alveolar epithelium. As hyperplasia of connective tissue occurs normally in the process of involution, it is probable that the fibrous induration generally found in senile lungs is part of this normal degeneration, while the slow accumulation of dust, acting as an irritant, plays but a small part in the proliferation of the connective tissue. Senile pneumokoniosis is almost invariably associated with atrophic emphysema, the two producing the typical atrophied lung of senility.

Symptoms.—The slow progressive pneumokoniosis of old age, which has not been aggravated by vocational dust inhalation,

gives no marked symptoms, except perhaps a hypertrophic bronchitis. The emphysematous symptoms which occasionally accompany it are due to the senile emphysema. The sputum is gray, tenacious and thick, containing leucocytes and cells from the bronchial and alveolar membranes with enclosed dust particles. Those suffering from the vocational forms of pneumokoniosis, like miners, knife grinders, glass and metal engravers, sand-blast operators, stone cutters, etc., rarely reach old age, as the dust particles which they inhale are sharp, cut into the tissues, cause necrosis and produce cavities.

Pneumokoniosis is sometimes called primary chronic interstitial pneumonia. The latter disease is generally localized and unilateral, and there is no discoloration of tissue. Septic infection frequently occurs and results in gangrene.

Treatment.—Pneumokoniosis is incurable. If there are distressing symptoms, such as dyspnea or excessive expectoration, the treatment must be directed to the emphysema or bronchitis which causes the symptoms. The patient should be removed from the smoky or dust-laden atmosphere, preferably to the seashore. If an aged person suffering from bronchitis moves from the clear atmosphere of the country, to a smoky city, distressing symptoms rapidly follow.

SENILE DEGENERATION OF THE ORAL CAVITY

The degenerative changes in the mouth and pharynx are not marked except by the loss of the teeth, yet this has a marked influence upon the health and welfare of the individual.

The teeth crack and break, their nerves and blood-vessels atrophy and with the atrophy of the alveolar process the teeth become loose and fall out. The mucous coating of the mouth becomes thin and pale, the glands atrophy, and likewise the salivary glands. The secretions are diminished but not altered. The change in the shape of the inferior maxilla is the most marked osseous senile change in the body. The muscles of mastication and deglutition waste and lose their tonicity. Owing to the loss of the teeth and the waste of muscles a decided effort must be made to approximate the gums and for this reason the jaws are never brought together except by a special effort of the will

although the lips may be closed easily. In some cases the openings of the salivary ducts are dilated permitting a dribbling of saliva into the mouth and if the lips are flaccid and not tightly closed the saliva drips out. As a result of these changes and of evident changes in the nerves and taste bulbs which have, however, never been demonstrated, profound changes occur in mastication and deglutition, in the appetite and in buccal digestion.

The *loss of the teeth* prevents mastication and necessitates a change in diet, the most important dietary change being the elimination of meat. While the loss of teeth can be repaired artificially, making the elimination of meat apparently unnecessary, this loss in the healthy aged person seems to keep pace with the senile degeneration of the stomach. Owing to degeneration of that organ meat digestion becomes more difficult and it would seem that the loss of teeth is really a natural provision to prevent excessive work for the senile stomach. *Diminished appetite* has probably the same purpose, lessening the amount of the ingesta in proportion to the needs of the system and to the diminished activity of the excretory organs. *Senile dysphagia* is part of the same natural provision to prevent overfeeding. The individual must make a sensible effort to swallow. Solids pass more readily than liquids; acid, sour, sharp, and acrid substances are swallowed more easily than alkaline, sweet and insipid substances. *Lessened thirst* is probably due to a dulling of the sensibilities of the nerve terminals and, like diminished appetite, is a compensatory arrangement to give less work to the heart and kidneys.

Nothing can be done for the dysphagia and nothing need be done for the other conditions except for the drivel of saliva. It is sometimes possible to instil a sense of neatness in the individual so that he will make an effort to control it. If this fails it may be possible to control it by local astringent applications to the mouths of the ducts. There is, however, danger of producing stenosis with complete drying and atrophy of the glands, a far more serious condition than the drivel. Atropia is contraindicated.

Glossodynia occurs occasionally and is probably due to the degeneration of the nerve terminals in the tongue. It is a paresthesia which does not yield to local treatment.

SENILE DEGENERATION OF THE STOMACH

While the anatomical changes of the stomach, due to the process of ageing, are marked, the functional impairment is slight, the demands made upon the senile organ being less. In senile tissue generally, the functions are performed less actively or differently from those of maturity but harmonious relations are maintained with allied organs and tissues, the functions of which are likewise impaired, thus maintaining the body in the state of functional equilibrium. This applies with special force to the stomach in which digestion is carried on sufficient for the needs of the aged organs while the same anatomical condition occurring in maturity would cause serious disturbance. There are several natural provisions for shielding the stomach in old age from excessive work. There is usually diminished appetite due to lessened need for food and to obtunded sense of hunger. Owing to the loss of teeth the aged individual cannot chew meat and he will take instead, eggs, milk and vegetable proteids which are more readily digestible. He has a distaste for flat and insipid foods and prefers salty and acid ones. The flat-tasting food is generally alkaline and as there is in old age a diminution in the quantity of hydrochloric acid, alkaline food interferes with digestion.

The prominent manifestations of senile degeneration of the stomach arise from atony and waste of the muscular fibers, diminution in the quantity of gastric juice and hydrochloric acid and atrophy of the mucous membrane. As a result of these senile changes we have atonicity and dilatation of the stomach, weakened propulsive power, prolonged retention of food, slow digestion of proteids and occasionally also insufficiency of the pylorus. Dietetic indiscretions will produce acute indigestion and gastric asthma and if prolonged, a chronic senile catarrh will result which may predispose to cancer.

Gastric Atonicity

Etiology.—Owing to the loss of tone of the muscular fibers the peristaltic waves are slower, the wave ring of circular fibers does not contract as powerfully, contractions of the cardiac portion are also slower and less powerful and food is not propelled as rapidly toward the pylorus nor is the food mixed as thoroughly

Myofibrosis (Chronic Myocarditis). (*Schmaus.*) × 150 diameters. *m*. Cardiac muscle-fibers. *b*, *b*. Newly formed fibrous connective tissue. This can often be demonstrated to be of different ages, and in the older parts calcareous change may have occurred.

with gastric juice. If food has been bolted without proper mastication, it may be retained in the stomach for many hours or it may pass into the duodenum unchanged.

While there is a natural loss of tone of muscular fiber in old age, this atonicity is increased by overfeeding, by ingestion of large quantities of liquids, by swallowing food in lumps, especially meats, and by a constant state of mental excitement such as worry, rage, etc.

Symptoms.—The symptoms of atonic dyspepsia are, a feeling of distress or bloating lasting for several hours after a meal; occasionally heartburn, cramps, nausea, belching of gas. Hyperacidity, which is common in younger individuals who then complain of acid eructations, is rare in the aged. There is usually tympany all over the stomach area and a splashing sound can be readily produced over an extensive area.

The most important physical sign of atonicity is the presence of food in the stomach seven hours after ingestion. This is determined by washing out the stomach, but the result may also be due to hour-glass contraction, pyloric obstruction or pylorospasm. Pyloric obstruction in the aged is generally caused by a growth; spasm is generally due to hyperacidity which is rare in the aged. The hour-glass contraction of the stomach is the result of a healed ulcer and contraction of the scar. It is rare in the aged. In pyloric obstruction due to growth the pylorus is palpable and peristaltic movements may be observed over the stomach proceeding from left to right toward the pylorus. In obstruction due to spasm the pylorus can be felt during the spasms but not in the intervals. Pyloric hypertrophy does not occur in the aged. The differential diagnosis between benign and malignant growths will be considered under Gastric Carcinoma.

Patients suffering from hour-glass contraction following ulcer rarely reach old age. Weinstein's test is to give the patient some raisins or figs. After two hours the stomach is washed out when the seeds will be found. The patient then walks around, shakes himself up, then lies down so as to shift the contents of the second chamber beyond the contraction to the anterior chamber. If the stomach is now again washed out seeds will be found that had gone into the second chamber and had been regurgitated into the first chamber.

In marked pyloric stenosis there is always a marked dilatation. In senile atony there is generally a slowly developing dilatation.

Treatment.—Normal senile atony will give no distress nor will it have any serious detrimental effect if dietetic regulations suitable for the aged are carried out. Owing to the normal diminution of gastric juice the proteid intake must be reduced, or if there is excessive waste of tissue and an increase in protein is necessary, pepsin and hydrochloric acid must be given with the meal. If the patient has bad teeth the food should be comminuted. Food should not be taken in shorter intervals than five or six hours. Fat should be excluded as far as possible and if given at all, it should not be used for frying foods. Butter is the best fat and may be used with bread. Vegetables can be taken but the greens should be avoided. Cabbage, lettuce, spinach, etc., have little nutritive value and entail much work upon the digestive system.

Fruits raw or stewed are good and spices and condiments may be used. Of beverages alcoholics are to be avoided.

If there is extreme atony, lavage once or twice a week may be tried; ordinarily it is not required. Medicinal treatment is seldom indicated. Occasional lapses from dietary rectitude must be corrected by a brisk cathartic or if there is much distress the stomach tube should be used.

Senile Dilatation of the Stomach

Etiology.—This is always due to atony and waste of the muscular fibers of the gastric wall. It occurs early in those who habitually overload the stomach especially among beer drinkers, and in those who have an early general arteriosclerosis. If these causes do not exist and the individual is careful about his food, the dilatation is slight and gives little or no sign of its presence.

Pathology.—The dilatation is usually moderate and involves the whole organ, unless the person has been an excessive eater when the fundus is greatly dilated. There may be marked gastroptosis. The walls of the stomach are thin but the pyloric orifice may be hypertrophied. The mucous membrane is thin and pale and the glands are atrophied.

Symptoms.—In mild cases there are no symptoms. Periodic vomiting, which is the most marked symptom of gastric dilatation in maturity, is rare in senility. Eructation of gas occurs frequently in beer drinkers and sometimes a small amount of fluid is brought up with the gas. Ordinarily there is a little belching of gas an hour or two after meals or if food is taken while the stomach still contains undigested food. Vomiting will occur if there is an excessive amount of food in the stomach, and some of it is decomposing, producing irritating toxins. It may also occur when there is some cause which would produce vomiting in other cases, such as shock, hemorrhage, cerebral anemia or an emetic. Clapotage can generally be elicited and gurgling can sometimes be felt over the pylorus. Unless there is extensive dilatation the physical signs are not marked. If the stomach is distended with gas, its outline may be made out by inspection and percussion and the fundus can be mapped out whenever there is a large amount of food present. The most accurate delineation of the stomach is obtained by radiography.

Treatment.—The most important indication in the treatment of senile dilatation is the regulation of food. The rule "little and often" is irrational and will make the condition worse. Owing to the slowed digestion, food should not be given in shorter intervals than five hours. Lavage is necessary only if too much or improper food has been taken. A binder will relieve the sense of weight following a meal but the benefit is psychic rather than physical. Medication is rarely indicated except perhaps to increase the tonicity of the abdominal walls. In such case strychnine in 1/60-grain doses may be given, care being taken to avoid excessive stimulation of the heart. Gastroptosis is sometimes relieved by an elastic abdominal binder. Surgical intervention is not required unless the gastroptosis is due to adhesions, bands or similar surgical conditions.

Pyloric Insufficiency

Pyloric insufficiency is a condition of the pylorus in which the orifice is not closed completely or with sufficient force, and partly digested food dribbles through. This, in the aged, is due

partly to waste of muscular fiber, partly to lessened innervation and partly to the weakened reflexes by which the pylorus opens and closes. These causes are directly traceable to senile degeneration of the muscular structure and of the plexus of Auerbach and to the diminished quantity of hydrochloric acid, which by irritating the gastric side of the sphincter causes it to open, and when irritating the duodenal side causes contraction and closing of the orifice. The only symptom which would indicate the presence of pyloric insufficiency is lientery, the stools containing particles of protein matter. (This might, however, occur if there is a deficiency of gastric juice and the diminished intestinal secretions are not sufficient to convert the proteids when there is a normal quantity of carbohydrates in the food.) This condition is sometimes detected after death. Treatment is the same as in gastric atonicity.

SENILE DEGENERATION OF THE INTESTINES

Pathology.—The degenerative changes in the intestines include atony and waste of the muscular fibers, thinning of the walls, with atrophy of the glands, the folds are smoothed out, and occasionally there are hernial protrusions and diverticulæ. Feces collect in the colon distending that portion of the intestines and the rectum and the wasted muscular fibers cannot overcome this distention. The colon consequently becomes dilated forming a pouch which may be 3 to 4 inches in diameter. There is lessened peristalsis, diminished intestinal secretion and lessened reflex irritability. As a result of these changes senile constipation is produced.

Senile Constipation

Etiology.—This is a symptom of senile degeneration of the intestines. The following causes contribute to this condition: (1) Diminished peristalsis due to atonicity of the intestinal walls; (2) diminished intestinal secretions due to atrophy of the intestinal glands; (3) diminished reflex irritability due to lessened innervation; (4) diminished bile supply thereby increasing the tendency to thickening of intestinal mucus and the formation of mucomembranes; (5) unsuitable food; (6) causes connected with gastric and duodenal digestion. Other causes of constipation as tumors, adhesions, hemorrhoids, viscerop-

Bramwell's "withered apple" heart. (Satterthwaite, *Med. Record*, May 14, 1910.)

tosis, habit, indigestion, etc., do not produce the condition here described which is simply a manifestation of senile degeneration.

Symptoms.—Senile constipation comes on slowly, the patient finding a gradually lessened desire for stool. If he is accustomed to go to stool at the same time daily, he must make a sensible effort to expel it and at times nothing will pass in spite of all straining. The stools are small and hard, dark and dry if they have been long retained, or light, clayey, if there is an abnormal deficiency of bile. Occasionally there is impaction of the colon with a canal through the impaction. When this is present the stools pass as small, hard balls. We can have a senile constipation with a daily evacuation; this seeming paradox occurs only when the feces are retained in the bowels beyond the normal period. In this condition the stools are hard, dark and dry and particles of food will be found that had been ingested two or three days before. In colonic or rectal impaction there is a sense of weight or fulness in the pelvis and a feeling or desire for a stool which does not pass upon straining.

Treatment.—In dealing with senile constipation we must take into consideration the various causes. There may be simply a dyschezia or inability to expel the feces from the colon and rectum. In this condition enemas are required and if the lower gut is impacted, the hard feces must first be softened by a prolonged high enema, of warm water containing a small amount of bicarbonate of soda. If this will not remove the mass it must be scooped out. It is sometimes possible to increase the expelling force by lowering the seat of the closet or raising the feet upon a foot stool. Cool rectal douches are often serviceable and in some cases rectal bougies containing ergotin and strychnine will increase the tonicity of the rectal walls. Astringent enemata will contract the rectal pouch, thereby lessening the tendency to impaction but they will not increase the expulsive power of the bowel. If the trouble lies solely in the lower bowel, cathartics are useless. In most cases the atonicity extends throughout the whole length of the intestines and then peristaltic stimulants are required. The most powerful of this class of drugs is a hypodermic injection of Eserin 1/100 gr. or aloes by mouth which increases peristaltic activity from the stomach to the sphincter-ani and produces a soft stool. The main objection to aloes is its tendency to cause congestion

of the lower bowel and to produce hemorrhoids. It gripes but this can be overcome by combining rhubarb with it. Belladonna which is usually added to overcome the griping effect is contraindicated as it lessens peristalsis. Other peristaltic stimulants are: cascara, senna, podophyllum, leptandrin and the bile salts. Rhubarb produces large, soft stools and it can be taken for years without causing habituation. It has a mild peristaltic effect and it increases the activity of the intestinal glands.

Leptandrin and podophyllum gripe and are inferior to senna, aloes, and cascara unless a hepatic stimulant is required at the same time. If there is a deficiency of bile it is better to supply this deficiency by using the bile salts instead of employing other hepatic stimulants, since the bile salts themselves also increase hepatic activity. Intestinal peristalsis is normally induced by the presence of indigestible portions of food, which act as a mild irritant to the bowels, and insufficiency of such matter lessens normal peristalsis. In old age there is a waste of the muscle fibers which produce peristalsis and there is probably a degeneration of nerve cells in the intestines. More powerful stimulation or irritation of the intestines is required to increase motility and this can sometimes be brought about by increasing the amount of food refuse and cellulose. As the ordinary foods containing much cellulose do not contain sufficient nutritional elements unless given in large amounts, a combination of readily digestible concentrated foods and substances containing little food matter and much cellulose is indicated. Spinach, cabbage, cauliflower, turnips, beets and carrots contain much cellulose. Rice and sago are constipating but other farinaceous foods may be taken. Fresh, tender, well-done meats may be taken, but meats that have been in cold storage should not be used, and this applies not only to constipation but to all senile conditions. Fresh boiled fish can be taken ad libitum. Whole wheat, graham and brown bread, and toast are good. Milk constipates many persons but buttermilk does not. Tea and spirituous liquors should be avoided. Pork, liver, mutton, all smoked and preserved meat and fish, cheese, pastry, sweets, eggs and nuts are objectionable. A glass of cold water at bedtime and a glass of hot water containing a pinch of salt or a teaspoonful of any of the cathartic salts will

help to flush the bowels. Large doses of salts should not be used as they withdraw water from the organism which the body cannot spare. Only in the obese are the salines of service.

Mechanical measures such as manual massage, massage by a mechanical roller, coarse vibration will sometimes relieve this condition. They act either by direct propulsion of intestinal contents or by producing a mild irritation and consequent peristalsis.

Among the most annoying concomitants of senile constipation are *flatulence* and *coliky pains* which are produced by the intestinal gases. These can be avoided by taking occasionally 5-grain doses of charcoal. As the loss of tonicity and diminished glandular activity in old age cannot be removed permanently a cure of senile constipation is impossible. Much can, however, be done by occasional stimulation of intestinal activity, by emptying the colon and rectum by means of enemata whenever there is a feeling of fulness, by adhering to a fixed hour for stool, using a low seat or foot rest to secure a more favorable position for defecation, and by careful control of the diet.

Atony of the Sphincter Ani

Atony of the anal sphincter is occasionally found in the aged as part of the general loss of tonicity of the intestines. The patient finds that he must make a sensible effort to close the sphincter after defecation and it requires an effort to keep it closed when there is desire for stool at an inopportune time. Small amounts of feces dribble out unconsciously and his clothes are constantly soiled, and when expelling flatus from the bowels he cannot control the passage of feces at the same time. If internal hemorrhoids exist they protrude from the anus. An effectual treatment of this condition is the occasional inunction of the sphincter with nutgall ointment to which 10 per cent. of ergotin is added. The base should be lanoline. Other astringents may be used but they may cause contraction of the rectal walls and constipation.

Anal Fissure

Anal fissure, while not a degenerative process, is frequently found in the aged, associated with atony of the sphincter. With the waste and atony of the muscular fibers, atrophy of

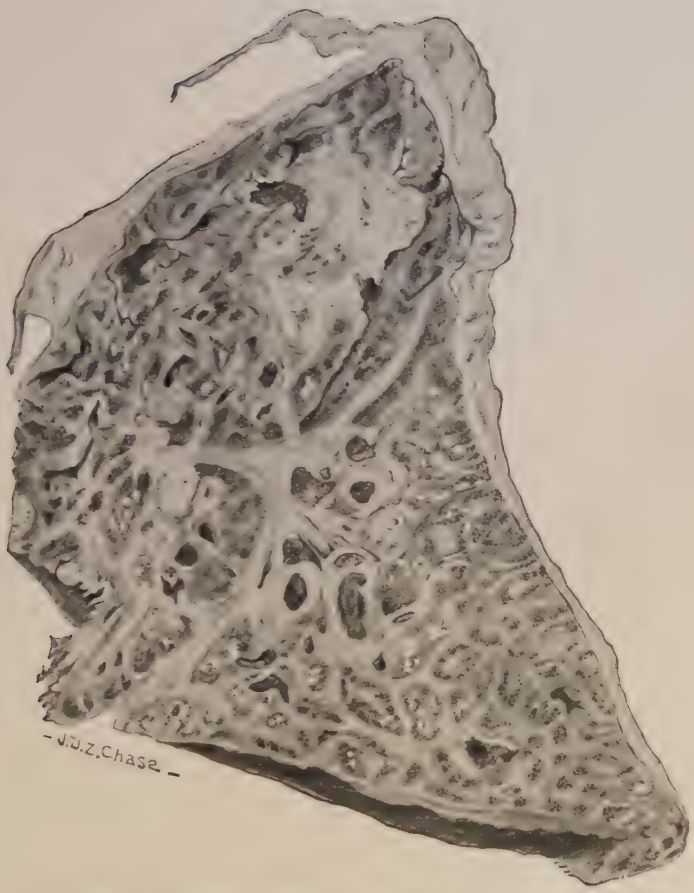
mucous membrane and changes in the skin immediately surrounding the anal orifice, slight causes will suffice to produce fissures in and about the sphincter. Hardened feces or the sudden expulsion of feces or scratching are the most frequent causes of this distressing lesion. The fissures themselves are generally very small, even microscopic, and when visible appear like short scratches about the sphincter. They may extend to the subcutaneous tissue and are then painful, otherwise they cause a pruritus which is aggravated by defecation. They do not heal readily as the passage of feces causes frequent irritation and the intolerable itching causes scratch lesions which may become eczematous or infected. A 2 per cent. cocaine solution will temporarily relieve the itching and a zinc ointment with a petrolatum base will prevent irritation and may effect a cure.

SENILE DEGENERATION OF THE LIVER

Senile degeneration of the liver is part of the general process of involution and is usually associated with and due to arteriosclerosis of the hepatic artery. Cases are occasionally found, however, in which the liver is degenerated while the artery is unimpaired.

Pathology.—The senile liver resembles the liver in the atrophic stage of cirrhosis. If there is no disturbance of the general circulation the organ is lighter in color than in maturity, but as there is usually some impairment of the circulation causing passive hyperemia the color is dark brown and dotted sometimes with yellowish spots of fat deposits. The organ is contracted, the acini are compressed through proliferation of the connective tissue and the capsule is thick, opaque and closely adherent to the body of the gland. The suspensory ligaments are weakened and this with a flaccid diaphragm, and with changes in the chest wall permits a ptosis of the organ with displacement to the left.

Symptoms.—The only marked symptom is the intestinal disturbance due to diminution of bile secretion, the stools being foul-smelling and light in color. If the bile is insufficient to emulsify the fat ingesta, fat globules will be found in the feces. Percussion elicits a diminution in the size of the organ and a probable ptosis with lateral displacement. It is sometimes possible to feel the upper border below the ribs while the tense



Pulmonary fibrosis, usually described as Chronic Interstitial Pneumonia, a condition frequently found in senile lungs. Illustration from Coplin's "Manual of Pathology."

Bands of fibrous tissue following course of interlobular septa and surrounding blood-vessels and bronchi. (*Landis, Fifth Ann. Rep. of Phipps Inst.*)

lower border can sometimes be felt below the umbilicus. It is not tender upon pressure and it gives none of the usual symptoms of cirrhosis. If jaundice is present it is invariably due to occlusion of the bile duct or to some pathological state of the gall-bladder.

Treatment.—Treatment is rarely indicated. The diminished amount of food taken by the aged requires a diminished amount of bile and it is only when this diminished quantity is insufficient —as evidenced by light-colored stools—that anything need be done to stimulate the functions of the organ. By far the best remedy in this condition is inspissated ox gall in 5-grain doses two or three times a day. This acts as a hepatic stimulant, causes a more fluid bile and at the same time supplies the deficiency of bile in the intestines. Other cholagogues like calomel, sodium succinate, sodium sulphate, sodium salicylate, benzoic acid and the many vegetable drugs may be given but none equals the natural bile salts.

SENILE DEGENERATION OF THE GALL-BLADDER

Senile degeneration of the gall-bladder is important on account of its interference with the secretion of bile and on account of secondary effects caused by retention of bile and formation of gall-stones.

Pathology.—The walls of the gall-bladder become thickened and rigid through thickening of the fibrous coat. This also diminishes the cavity of the gall-bladder and a contraction of the walls causes a diminution in the size of the organ. Occasionally there are lime deposits in the walls and a layer of inspissated cholesterin lining the cavity has been reported. The neck is contracted, the lumen of the cystic duct is diminished and in rare cases it is entirely obliterated. The common duct is usually dilated.

The amount of bile is diminished and it contains more cholesterin than in maturity, the secretion becomes jelly-like and frequently hardens, forming gall-stones. These may give rise to cholecystitis and the usual symptoms of gall-stones.

Symptoms.—Degeneration of the gall-bladder gives no symptoms unless the gall-stones increase in size or number so as to cause local inflammation, or move toward the duodenum

when they may produce the familiar symptoms of cholelithiasis. Complete occlusion of the cystic duct may occur without marked symptoms, the bile flowing from the liver through the hepatic and common ducts to the duodenum. If, however, the common duct is occluded there is jaundice and the intestinal symptoms of deficient bile secretion are produced, such as clayey, foul-smelling stools containing fat globules. It is often impossible to determine whether the deficiency of bile in the intestines is due to the liver, cyst or ducts as it may be caused by a diminished formation, a change in the character of the secretion or an obstruction to the flow. If there is obstruction and consequent jaundice, the fault lies in the ducts. A change in character may arise in the liver but is more probably due to prolonged retention in the gall-bladder. This may give rise to occasional colicky pains but the diagnosis cannot be positively made until the symptoms of cholecystitis or cholelithiasis appear.

Treatment.—The only medical treatment applicable to degeneration of the gall-bladder has for its purpose the production of a more fluid and a more copious flow of bile, as has been recommended under the treatment of degeneration of the liver. This has no effect upon the degeneration itself, but it enable the organ to perform its functions more readily and prevents irritation and inflammation. If cholecystitis or cholelithiasis occurs, surgical intervention is necessary.

SENILE DEGENERATION OF THE KIDNEY

The senile contracted kidney is the physiological kidney of old age and is mentioned here only because pathologists frequently report the finding at autopsies of chronic interstitial nephritis that gave no symptoms during life.

The senile anatomical changes have been described. They differ from the pathological changes found in nephritis in the absence of hyaline and fatty degeneration and of cloudy swelling in the tufts and vessels. The epithelium of the tubules shows no change and the tubules are clear and free from the granular detritus which generally obstructs the tubules in nephritis.

Symptoms.—The only symptoms of senile contracted kidney are a trace of albumin in the urine which may persist during life and a diminished secretion of urine. The specific gravity

of the urine is normal or but slightly decreased, the urates are considerably diminished and calcium salts also are generally diminished. The diminution of the urates and calcium salts is, however, not due to the kidney changes but to metabolic changes and only the diminished quantity of urine of normal or slightly decreased specific gravity and a persistent albuminuria *without casts* are indicative of senile contracted kidney (see Chronic Interstitial Nephritis).

Treatment.—Nothing need be done unless the amount of urine is markedly diminished when drinking large quantities of alkaline mineral water will be found effective. Other diuretics are rarely required.

SENILE DEGENERATION OF THE BLADDER

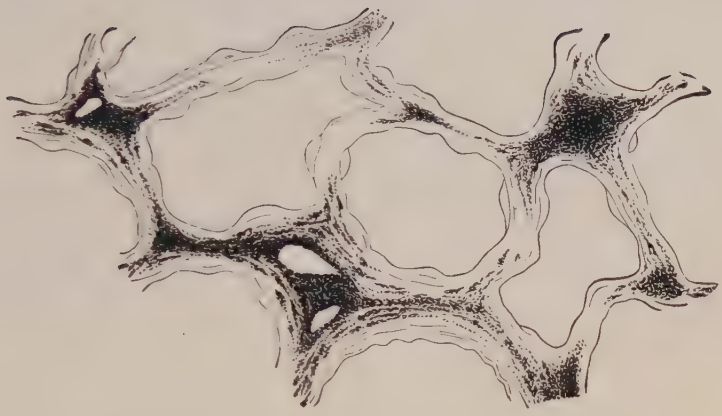
Etiology.—While dilatation of the bladder through atony of the muscular coat is part of the normal process of involution the condition is aggravated through the retention of urine. Thus one of the many vicious circles of senility is produced. The atonicity of the organs permits retention of urine which further distends the bladder, further stretching and weakening the muscular coat, and this in turn permits greater retention and distention. A frequent cause of retention of urine is a hypertrophied prostate which obstructs the free passage of urine. In some cases the diminished sensitiveness and irritability of the organ makes the need for micturition less strongly felt and the aged individual neglects it.

Pathology.—The pathology is an exaggeration of the normal senile changes. There is atony and waste of the longitudinal muscular fibers and of most of the circular fibers. Some of the latter degenerate into fibrous bands which contract, and the weakened wall bulging out between these contracted bands forms rugæ, pockets and pouches. The mucous membrane becomes atrophied, the whole organ is dilated and its contractility is lessened. In rare cases the contractions produced by the fibrous bands may be so numerous or extensive as to diminish the capacity of the organ. There is usually atony of the sphincter due to waste of the muscular fibers and sometimes to pressure of an enlarged prostate.

Symptoms.—The earliest symptom of degeneration of the bladder is a diminution in the expulsive power of the organ. The patient cannot send the stream as far as formerly. Then he finds that it requires a sensible effort to void the urine, that it takes a few moments of straining before the flow appears and instead of coming out with force, it is sluggish. Later the stream is small and drops from the meatus. After the bladder has become dilated there is retention of urine and in addition to the other symptoms there is a frequent desire to urinate, especially at night, and if there is atony of the sphincter the patient may wet the bed. There is generally a sense of weight in the pelvis, relieved after the bladder is emptied. Dribbling is always due to atony of the sphincter. Occasionally there is constant dribbling, the urine flowing away as soon as it enters the bladder from the ureters. If there is marked atonicity of the bladder and sphincter there may be retention and incontinence.

Diagnosis.—The diagnosis of dilatation of the bladder and atony of the sphincter are readily made but care must be taken to eliminate other factors which may cause these symptoms. There may be retention without atonicity—due to stone, tumor, hypertrophied prostate or an old stricture. In such case there will be difficulty in starting the flow, but after it is started it comes out with force whether coming naturally or if drawn off with a catheter.

If due to stone the trouble is worse when the patient is much on his feet; if due to enlarged prostate the difficulty is more pronounced at night or in the early morning. A positive diagnosis is made by means of rectal examination, sound and cystoscope. This also applies to growths. Dribbling or passing a few drops of urine after urination is apparently completed is evidence of atony of the sphincter. If after catheterization the patient is laid upon his back, then turned upon his face and made to arise, the desire to urinate or the presence of more urine upon an immediate recatheterization shows the presence of retained urine in the vesical pouches. These may be minute and hold not more than a drop or two in each pouch but when these drops are long retained they decompose, cause local irritation and inflammation. A distended bladder can readily be made out by palpation and percussion and confirmed by the



Section of the Lung, Pneumoconiosis. (*Rindfleisch.*) The deposited pigment is shown in the connective tissue of the vesicular wall.

Dilated stomach. Combined displacement and dilatation of lesser degree. (From Greene after Riegel.)

catheter. Without this confirmation it may be mistaken for ascites. Occasionally the insertion of the catheter will excite urethral spasm which may be mistaken for some other form of obstruction. An injection of a 2 per cent. solution of cocaine in warm water will relieve spasm but not organic obstruction. The character of the urine gives us little information. A retention urine is ammoniacal, due to decomposition, and is turbid. If the turbidity is not cleared up by heat it is due to bacteria; but bacterial urine is frequently found in the aged without discernible cause or ill effects. Pus and blood in the urine may be due to vesical disease but they do not occur in uncomplicated senile degeneration.

Treatment.—In the treatment of senile bladder affections the first indication is to empty the bladder and secure an evacuation of it at intervals of not more than eight to twelve hours. The patient should be impressed with the necessity of attending to the demand for evacuation of the bladder without delay and if he feels that he has not passed all, he should get on his knees and press the edge of the vessel against the perineum. If catheterization becomes necessary the patient should be taught how to use it and how to sterilize it. The sterilization must be repeatedly insisted upon until it becomes a habit, as the aged become forgetful and careless, the few drops of urine remaining in the catheter decompose and with the next insertion bacteria are introduced into the bladder. Internal medication has two objects, the prevention of decomposition and the increase of tonicity. If there are other pathological conditions present these require treatment apart from the treatment for atonicity. Decomposition is prevented by the use of hexamethalenetetramin which appears on the market under various trade names, urotropin, formin, cystogen, uritone, etc. The dose is 5 grains twice daily, always to be given in solution. No other urinary antiseptic approaches this in efficacy. To increase the tonicity of the organ, strychnine and electricity will be found beneficial. Belladonna which is almost a specific in incontinence of urine in childhood aggravates the senile atony of the sphincter. If there is no arteriosclerosis ergot can be given in doses of 15 minims of the fluid extract three times a day. If there is arteriosclerosis with high blood pressure its powerful vasoconstrictor effect may cause cerebral trouble.

Aloes has been recommended on account of its property to stimulate peristalsis thereby increasing the activity of the muscle fibers. This action is directed more particularly to the lower intestinal tract and it has little if any effect upon the bladder. The injection of astringents and mild silver solutions have sometimes a temporary beneficial effect. A single distention will undo all the good that may have been achieved by treatment.

SENILE DEGENERATION OF MALE GENITALS

Senile Impotence

The male reproductive organs undergo profound anatomical changes, out of all proportion to the functional changes that occur in old age.

Pathology.—The *testes* are atrophied, the fibrous coat is thickened, there is a proliferation of connective tissue throughout the gland, compressing the lobules and their seminiferous tubes, some of which are completely obliterated, while in others the lumen is compressed and occluded. The *vas deferens* is hardened and thickened, its caliber is diminished and the *seminal vesicles* are atrophied. The *semen* becomes more fluid and transparent, while in some pigment is deposited giving the semen a brown color, and the spermatozoa are diminished in number but their functional activity is not impaired. Active semen has been found in the tenth decade of life.

The *penis* is shrunken, the glans hardens and the whole organ becomes less sensitive. The skin of the penis and scrotum undergoes the same changes that occur in other parts of the body and in addition the whole genital region becomes pigmented and there is often a fetid bromidrosis.

Functional Changes.—Diminution both in sexual desire and power of erection is generally noticed during the fifth or sixth decade and these are the principal manifestations of the male critical period; in many cases, however, there is apparently little or no loss of functional activity. Where diminishing libido and potentia proceed together the impairment may not be noticed, since neither mental nor physical distress is produced. It is only when attention is called to the lessened functional powers—as by marriage with an erotic woman—that the condi-

tion is recognized. In some cases the desire remains after the power of erection has waned, a condition frequently found in confirmed masturbators.

Treatment.—The treatment of senile impotence depends upon the general physical condition of the individual and attending circumstances. In cases where there is a gradual diminution in erectile power and a diminution in desire nothing need be done unless marriage is contemplated. We must bear in mind that the intense mental and physical exertion during coitus, and the succeeding depressing reaction are detrimental to the aged individual and if there is uncompensated heart disease a fatal result may ensue. Where the desire remains but the power is lost it is often difficult to decide whether aphrodisiac or anaphrodisiac measures shall be employed. The object of treatment in such cases is to diminish the libido and at the same time slightly increase the potentia in order to produce a favorable psychic effect. The patient's wish is to have the power of erection restored and he will not willingly follow any treatment which will lessen the desire. It will therefore be necessary to resort to anaphrodisiac drugs such as bromides, monobromated camphor, valerian and lupidin, and at the same time use local stimulants, warm applications, massage and electricity. The water applications and massage have only a momentary effect, still sufficient to satisfy the patient. As the frequency and the intensity of the libido diminish under above drug treatment, local stimulation can be lessened and finally omitted entirely. Electrical stimulation by means of the faradic current is more permanent but it should not be employed unless the other measures fail or a prolonged effect is desired. Local irritation, as from a hypertrophied prostate, may keep up the hypererosis. Lascivious literature, pornographs, the goading and teasing of thoughtless friends, all tend to keep the mind directed to the impaired function. The removal of every source and form of erotic stimulation is necessary for success. If aphrodisiac treatment is desired, the above-mentioned local measures should be employed and in addition the ordinary aphrodisiac remedies may be given internally. Ergot which is probably the best drug for functional impotence cannot be used in senile cases on account of its vasoconstrictor effect. Phosphorus or zinc phosphide with

nux vomica are reliable aphrodisiacs but the action is not permanent and constantly increasing doses must be given. Sandalwood oil in 10-minim doses three times a day is sometimes effective. Damiana and muirapuama are of little use in the aged while yohimbin, recommended as an aphrodisiac, is an irritant to the genital organs, the irritation producing the erection. A lymph compound consisting of lymph, lymphatic gland extract, brain and cord extracts from goats and orchitic fluid from bulls has been reported effective in some cases.

Psychic measures usually give immediate and often lasting results, marriage with a young person being sometimes followed by happy results without other treatment. Association with young persons, flattery by a person of the opposite sex, sensuous pleasures, all tend to bring about the desired effect.

Senile satyriasis, inordinate erotic desire, occurs occasionally in the aged. The bromides in large doses will cure this condition. If occurring as a recrudescence after years of quiescence, it is a symptom of senile dementia. It then usually appears during the senile climacteric and may lead to acts of violence. Bromides and narcotics are required. Threats are useless.

SENILE DEGENERATION OF THE PROSTATE

The senile changes in the prostate are hypertrophy and atrophy of the gland. Hypertrophy, which is found in about one-third of all cases, is an anomaly in senile involution as it is the only case presenting an increase of glandular tissue as a senile change. Many theories have been advanced for this peculiarity; none is satisfactory. A rational explanation may be found in the theory of tissue-cell evolution advanced in this work.

Pathology.—The hypertrophy assumes various forms. Sometimes the whole gland is enlarged, occasionally it is limited to one or both lateral lobes, more often the middle lobe is larger than the others. In most cases the hypertrophy consists of glandular, muscular and fibrous elements, the latter two predominating. Occasionally the glandular element is greater, producing a soft hypertrophy. The mass is generally unsymmetrical, may reach the size of a hen's egg and contains numerous small fibrous tumors, which in connection with

Fissure in ano. (From Gant's "Constipation.")

prostatic concretions frequently block up the ducts. If the hypertrophy is lateral it twists the prostatic portion of the urethra and if central it flattens and compresses the canal.

Symptoms.—While cases have been recorded in which prostatic hypertrophies have been found which gave no symptoms during life this condition usually gives early evidence of its presence. The old man finds that he must urinate more frequently, especially at night, that he must make a sensible effort to start the flow and that it comes in a small stream without any force behind it. Later on, there is a feeling of uneasiness or of dissatisfaction as if he had not been able to completely empty the bladder and he wants to pass a little more. There is always an amount of residual urine in the bladder which decomposes and may produce cystitis. After a time there is retention of urine with dilatation of the bladder, still later the atony of the sphincter induced by the senile waste of the muscle fibers or by paralysis caused by pressure, permits dribbling and we have retention with incontinence. There is now atony and dilatation of the badder with retention of decomposing urine, atony or paralysis of the sphincter, the lumen of the upper part of the urethra being diminished and perhaps twisted, and owing to the size of the gland it is forced upward carrying the neck of the bladder with it. Urination is now impossible without the catheter and catheterization must be performed two or three times a day. A frequent complication is septic bacterial infection produced by the catheter. Urosepsis from this source leads to a train of septic inflammations beginning with urethritis and cystitis and involving allied organs and tissues until pyelitis and exhaustion terminate life. The diagnosis of prostatic hypertrophy may be made by rectal examination in the knee-chest position. The only conditions which present a growth or tumor in the locality of the prostate are cancer and stone. Cancer gives the symptoms of cachexia, hematuria and pain. Calculus is movable and its diagnosis can be confirmed by the searcher and cystoscope.

Treatment.—The excellent results achieved in recent years by partial and total prostatectomy and the unsatisfactory results of medical treatment have removed prostatic hypertrophy from the field of the physician to that of the surgeon. Shall it be a complete or a partial removal of the mass, shall it

be extirpation or enucleation, suprapubic or perineal, shall it be performed early or late? Each has its advocates and opponents and each has been successful. Every surgeon has his favorite method of operation and judging from results there is little to choose between them. Perineal enucleation seems to have a lower mortality but more frequent unfavorable after-effects, such as fistulæ, strictures, incontinence, etc. As for the time when the operation shall be performed some advise it before the catheterization habit has been formed, others favor it when the catheter fails to remove the residual urine, others again will not operate except as a last resort. Bottini's galvano-cautery operation has not given the favorable results in the hands of his followers that he has reported in his own cases. Local applications, formerly much in vogue, have fallen into disuse. The only drugs from which any improvement has been reported are iodine, silver and mercury, yet real benefit from either has been rarely recorded. Straightening and stretching the urethral canal by means of sounds has relieved the dysuria but has no effect upon the prostate. If catheterization becomes necessary the old man should be taught how to use the catheter and how to sterilize it. He generally becomes more expert in the introduction than the physician himself, but there is some danger that in his hands it will lead to sexual perversions. The family should look after the sterilization of the instrument and this should be performed immediately before and after its use.

Atrophy of the prostate is the form of degeneration that is analogous to the senile degeneration in other glandular organs. It occurs in about 10 per cent. of the cases. It is only of importance when the proliferated fibrous bands constrict the prostatic portion of the urethra and interfere with the free flow of urine. In that case sounds should be used to dilate that portion of the canal while the catheter is used to draw off the urine. In extreme cases surgical measures must be resorted to to relieve the ischuria.

SENILE DEGENERATION OF THE FEMALE GENITAL ORGANS

The senile degeneration of the female genital organs generally occurs during the middle or close of the fifth decade. The anatomical and physiological changes are well marked, proceed

rapidly and are accompanied by constitutional symptoms and often by pronounced changes in the entire organism. The external genitals atrophy, the labia are shrunken, flabby and do not completely close the vulvar aperture leaving the vaginal orifice exposed. The skin is dark, wrinkled and leathery; the hair thin and gray. The vagina is wrinkled, dry, easily dilated and owing to the loss of tonicity of its walls, the latter fall together. Occasionally the vagina shrinks, its caliber is diminished and there is a progressive atresia. The uterus undergoes atrophy with marked histological changes. The atrophy proceeds rapidly during the period of the menopause then continues slowly during the rest of life, finally falling below the weight and size of the uterus at puberty. The walls contract, diminishing and occasionally obliterating the cavity. The cervix becomes elongated. In some cases annular or partial constrictions of the walls of the cervix and body cause a series of dilatations or completely enclosed cavities. The mucous membrane is smooth, but occasionally it is covered with minute nodules, the remnants of glands. The external coat of the uterus is usually tough, leathery and wrinkled. The muscular coats atrophy, there is a hyperplasia of fibrous connective tissue and the elastic fibers gradually disappear. The blood-vessels degenerate and may become obliterated. The *Fallopian tubes* become obliterated and appear as strings on the border of the broad ligaments. The *ovaries* atrophy, become hard, dense, sometimes containing calcareous deposits, rarely calcareous incrustations. The ovaries shrivel and have a rough knobbed appearance after the menopause but late in life they become smooth and flattened. The histological changes are similar to the changes in other solid organs, a waste of muscle and elastic tissue and a hyperplasia of connective tissue. The Graffian follicles degenerate into minute nodules or disappear leaving cavities.

The essential physiological change is the cessation of menstruation. Incidental thereto is atrophy of the mammary glands with the disappearance of milk and followed generally by neuroses and sometimes psychoses.

While the menopause and the discomforts incident thereto are physiological, the latter may be so pronounced as to require medical intervention.

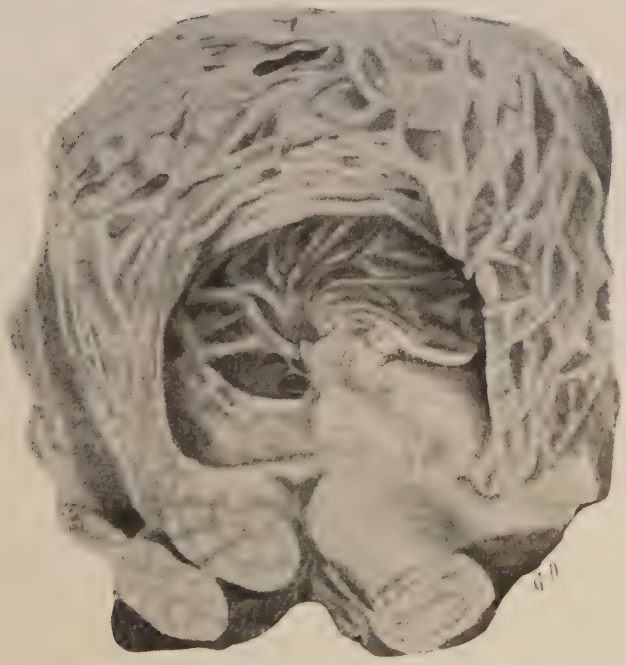
The menses may cease suddenly or they may appear at irregular intervals before their final disappearance. Nothing can or need be done to bring them on although women fearing that delayed menstruation means pregnancy, will often insist upon doing something to cause their reappearance. A persistent sanguineous discharge between the menstrual periods is indicative of a grave uterine disorder, frequently a malignant growth. It may be due to inflammation or traumatism produced by instruments used to produce abortion.

The uncomfortable sensations, flushing of the face, a feeling of heaviness or uneasiness, occasional disinclination for work and at other times excessive activity, irritability, etc., that accompany the menopause can often be relieved by the use of dried corpora lutea given in 10-grain doses several times a day. This should be discontinued as soon as its therapeutic effect is produced and resumed when the disagreeable sensations return. The flushes and headache can be speedily relieved by a hot foot bath. An intolerable vulvar pruritus is sometimes present and can be temporarily relieved by a 2 per cent. cocaine solution or ointment.

The neuroses and psychoses that accompany the menopause are often intractable but they usually disappear at the completion of the period. Irritability with occasional violent, uncontrollable outbursts of temper, and hysteria may occur. These are best treated by bromides and chloral. Hyoscine given hypodermically in 1/100-grain doses will generally quiet the patient during the violent attack. In many cases, however, the outburst of temper lasts but a few moments and is followed by hysterical tears or by a sound sleep and no treatment is required. Women sometimes know when these attacks will come on, they know or feel that they will have a bad day and while some will try to fight off the approaching feeling of irritability, others give vent to their feelings and refuse every effort to relieve them. The treatment of these cases depends to a great extent upon the temperament of the individual and psychic measures are often more effective than drugs. The menopause usually produces a profound psychic depression in childless women. Moral suasion is occasionally effective in these cases but drugs are useless except to produce a momentary stimulation.

Atresia of the senile vagina seldom produces much discomfort

Senile Contracted Kidney. (Pic and Bonnamour.)



Senile Bladder showing also hypertrophied prostate. (Pic and Bonnamour.)

and this condition may pass unnoticed unless discovered accidentally. There may be a local or annular constriction, more often the diameter of the canal is diminished throughout its length. In a few cases the constriction is sufficient to interfere with the free discharge of the uterine and vaginal secretions, and in rare cases the occlusion is complete, preventing the discharge entirely. There is very little secretion from these parts in the aged, virtually none when the woman is in the recumbent position. When walking a slight mucous discharge appears. The retention of this discharge will produce a catarrhal vaginitis and may cause a metritis; occasionally it becomes mucopurulent and may then lead to local septic infection. The treatment depends upon the extent of the atresia. Omitting the atresias due to cicatrical tissue and other surgical growths, conditions which are not considered here, atresia of the vagina in the aged seldom requires treatment. In some cases astringent injections will serve to diminish the discharge and it is often possible to dilate the canal with the finger. For localized constriction a tampon with boroglyceride will usually be effectual. In the rare cases of complete occlusion surgical intervention is necessary.

SENILE DEGENERATION OF THE DUCTLESS GLANDS

Neither the anatomical nor the physiological senile changes in these glands are well understood. While they generally undergo atrophy, hypertrophy is occasionally found, yet there does not seem to be any marked perversion of function in either case. Pic says, the senile changes explain the lessened resistance to infection and intoxication, yet the aged have apparently increased resistance to many forms of infection since infectious diseases except erysipelas are relatively less frequent in them and when they do occur they are usually milder, and autointoxications are also borne better. Lorand ascribes the senile changes mainly to atrophy of the thyroid gland yet thyroid medication will not retard the changes. From our limited knowledge of the internal secretions and their intimate interrelations, increasing or diminishing the work of each other, it is probable that so long as they maintain their functional relations to each other the harmonious relations of the whole organism are maintained. This of course presupposes that other organs are not impaired.

Atrophy of the ductless glands lessens their secretions and consequently their functional activity but counteracting influences, as the vasomotor action of the suprarenals and thyroid, prevent a disturbance in their action. In some cases excessive atrophy in one gland is compensated by increased activity in others as may occur when the spleen is greatly atrophied and the lymphatic glands become enlarged. Lacking, however, more definte knowledge of the functions of the ductless glands and especially of their functions when they have undergone senile degeneration, work in this direction must be based upon hypothetical assumptions and animal experimentation which leave a wide margin for faulty conclusions.

Spleen.—The spleen is generally atrophied, and its weight is greatly reduced, weighing as little as 40 grams instead of 100 to 150 grams, the normal weight in maturity. The Malpighian corpuscles are correspondingly atrophied, the fibrous tissue is increased and the walls of the arterioles are considerably thickened, while the arterioles themselves may be completely obliterated through pressure or constriction produced by fibrous bands. The color ranges from bright red to red brown. The capsule is closely adherent, thick, rough, opaque, the organ presenting the appearance of a withered apple. In some cases the spleen is found hypertrophied but in these cases there can usually be obtained the history of a disease with which splenic hypertrophy is associated.

In cases of extreme atrophy the spleen consists of a mass of fibrous trabeculi in which particles of spleen substance are entrapped, and in which most of the blood-vessels are obliterated. Notwithstanding these profound changes there is apparently no change in the functions of the organ. It may be, however, that there is a compensating increase in the functions of the lymphatic glands, as total extirpation of the spleen in maturity is followed by hypertrophy of the lymphatic glands. The lymphatics are, however, usually atrophied in the aged. It is impossible to harmonize the generally accepted views concerning the functions of the spleen and lymphatics with the conditions that exist in the aged. The number and proportion of leucocytes are not diminished, nor is the character of the blood materially altered. Malaria in the aged follows a milder course than in maturity and the plasmodium which is believed to be

destroyed in the spleen, disappears under treatment as rapidly as in maturity, though the spleen be extremely atrophied.

No pathological condition and no disease symptoms can be traced to senile degeneration of the spleen.

Thyroid Gland.—The thyroid gland is atrophied in the aged. There is a hyperplasia of the fibrous structure which compresses the alveoli, causing them to atrophy, thereby making the whole organ denser. These changes proceed very slowly and the organ is normally active to a very old age. There is a vast difference of opinion respecting the physiological functions of the thyroid in the aged. While Pelliet declares that the thyroid is one of the organs which retains its physiological activity longest in senility, Leopold Levi and Lorand ascribe to it many or most of the senile changes. Indeed Lorand expresses the belief that old age can be deferred by preventing senile atrophy of the thyroid or by replacing the waste of the organ.

According to Horsley, the senile cachexia presents a remarkable resemblance to myxedema, which is due to a deficiency of thyroid secretion, while those aged persons who do not present this cachexia have active, healthy thyroid glands.

While the thyroid gland has a profound influence upon nutrition and a diminution of its secretion is the cause of cretinism and myxedema, it is extremely doubtful whether any of the normal senile changes are dependent upon the state of that organ. Some of the changes noted in myxedema are seen in senility but these are probably incidental to similar degenerative results, not causes.

Edmunds found that if calcium was administered to animals in whom the thyroid and parathyroid were excised, the usual rapid death following complete thyroidectomy was prevented. As there is an increased proportion of lime salts in senility, this may explain why functional activity is but slightly disturbed in senile atrophy of the gland. Our knowledge of the functions of the gland is still imperfect and no definite statements can be made as to the character of the functional changes in the aged.

Suprarenal Glands.—The suprarenal gland is generally atrophied; Salrazes and Hushot who examined this organ in a number of aged persons, found it, however, often hypertrophied. There are two antagonistic senile processes, a sclerosis of the vessels and an increase in glandular tissue, due to fat deposits

in the cells of the cortical layer. In the lower reticulated layer the cells are surcharged with pigment. Contradictory views are held concerning the functional changes of the suprarenals, some authorities believing that there is a hypersecretion producing or aiding in the production of atheroma, high blood pressure, pancreatic degeneration, abnormal pigment deposits, etc., others denying that the senile organs play any such rôle in the senile processes. Since it is uncertain just what rôle the senile suprarenal glands play in the organism, we cannot say what perversion of function or what symptoms are produced.

SENILE DEGENERATION OF THE SKIN

The senile changes in the skin have been described in the chapter on Anatomical Changes in Old Age. It is, however, sometimes impossible to say whether some of these changes are physiological or pathological, due to the process of involution, to malnutrition following arteriosclerosis, to dyscrasias, to local lesions, or are natural and normal conditions under certain circumstances not connected with age. Shall we say the weather-beaten skin of the sailor is pathological because it is dryer, coarser, rougher, more wrinkled and more pigmented than the skin of the city dweller, or is it a senile condition because the senile exposed skin gradually assumes the same characteristic, or is it a normal condition of maturity? The skin of the aged is dry and it is generally possible to rub off fine white scales of dried epithelium. Is this pityriasis tabescentium a disease? Some of of the manifestations of senile changes like pruritus, pigmentation, etc., are distressing or annoying, while some like bromidrosis may exist through life, obvious to everyone except to the individual himself.

There are various perversions of the normal senile changes in the skin, some occurring locally, others being scattered all over the body. The latter are really dyscrasias or due to changes in the blood vessels. (These will be taken up in the second group.) Owing to the changes in the skin almost all dermatoses occurring in the aged are modified, some presenting marked differences between those of maturity and those of senility.

Some of the manifestations of the senile changes in the skin

are annoying on account of their appearance. Such are folds and wrinkles, pigment deposits, the changes in the hair, etc. The aged are often more concerned about these than about the more important pathological conditions and these are generally the most difficult to treat. *Pruritus* though usually included among the diseases of the skin is dealt with under the degeneration of the nerves as it is probably a symptom of degeneration of the nerve terminals.

Wrinkles are usually the earliest of the senile changes and are due to waste of muscular fibers leaving layers of fat. It is impossible to replace these fibers and if the fat also wastes the skin hangs in folds. Massage will improve the surface circulation and stimulate the remaining muscular fibers; it might, however, cause absorption of the fat and leave the skin in folds. This can be avoided by inunction with an animal fat, either cream, sweet butter, lard or lanoline, and mild massage. The skin should be massaged across the wrinkles, not along the wrinkles.

Florida water, glycerin, bay rum and washes, ointments, and other toilet preparations containing alcohol or other desiccants, are harmful.

Pigment deposits upon the face, neck or hands are unsightly but there is no safe and certain method of removing them. There are usually extensive areas of pigmentation about the genitals and inner surface of the thighs, sometimes upon the chest and frequently upon the back and arms. Pigment deposits on the face, neck and hand are generally in patches and spots. The usual treatment for chloasma patches and freckles applicable in younger persons will not avail in senility as the drugs employed are either irritants or deoxidizing bleaches which act as irritants, and owing to poor surface circulation the injury done to the skin by them is not readily repaired. Even slight surface irritation is liable to cause an ulcer or gangrene.

The senile skin requires special care to avoid irritation and to render it presentable. Soaps containing large amounts of alkali or glycerine are injurious, likewise lotions and applications containing alcohol. Inunction with animal fats is benefical but mineral fat is worthless and may be injurious as it is not absorbed and blocks the pores. Face powders and talcum are positively injurious.

Senile Alopecia

Alopecia or baldness is a symptom, not a disease, although the pathological condition to which it is due is not always evident. There is a primary or idiopathic form including congenital, premature and senile alopecia and a secondary form due either to a local disease as seborrhea or to a general cause as syphilis.

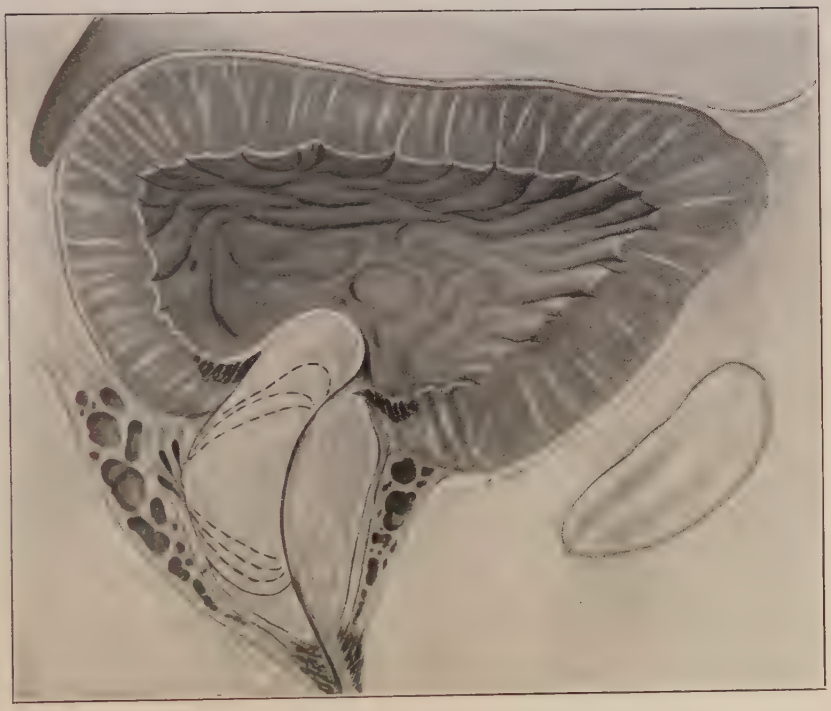
Senile alopecia may appear in the fifth decade but is usually first noticed about the end of the sixth decade and where there has been little falling out of the hair before the senile climacteric it falls out rapidly during or immediatly following this time.

Pathology.—The corium which forms the walls of the hair follicles atrophies and the mouth of the follicle becomes filled with epidermal débris. The sebaceous glands atrophy and the hair consequently becomes ill-nourished and dry. While the follicular walls can still hold the roots in place the dry hair becomes thin and breaks at the point of exit from the skin. When the follicular walls become so changed that they cannot hold the roots the hairs fall out on brushing or combing.

Symptoms.—In senile alopecia the hair about the central point of the scalp back of the crown becomes thin and the hairs break off at the point of exit from the scalp. The breaking of the hair later occurs all over the scalp, not in patches as in alopecia areata, but isolated hairs break everywhere. Owing to the atrophy of the corium the roots become loose, fine dust and epithelial débris work their way under the roots and push them up and brushing or combing dislodges them. The hair falls out first about the center of the scalp, the denuded surface gradually extending forward, and slowly backward, a thin border of hair being left at the sides and back. In some cases the hair first breaks over the temples then the rest of the scalp is involved.

Treatment.—The treatment of senile alopecia has been unsatisfactory because the measures employed were based upon unscientific empiricisin. Baldness is the end result of many different conditions and the treatment must consider the primary or causative condition. Neglect to discover this underlying cause is responsible for the general failure of any certain line of treatment which had been successful in a few cases.

When the hair falls out during a fever it will grow again without any treatment. If due to parasitic sycosis, treatment insti-



Progressive stages in Prostatic Hypertrophy; Semidiagrammatic. (Squier, *Medical Record*, Feb. 3, 1912.)

tuted before the follicles and roots are destroyed will save the roots and the hair will grow; if the follicles or roots are destroyed nothing will avail. In seborrheic dermatitis after the dandruff is cured, the growth of hair can be stimulated providing the roots and follicles are not destroyed. In senile alopecia the success of treatment depends upon the condition of the skin, follicles, roots and sebaceous glands. The sebaceous glands are generally atrophied but the diminished sebum can be replaced by an animal oil or fat. Petrolatum is *not* an animal fat and its employment is harmful. The mouths of the follicles should be cleaned out by washing the scalp with a 2 per cent. solution of biborate of soda. After the head has been cleaned a mild stimulant should be used. The best for this purpose is tincture of cantharides, capsicum or jaborandi, using 1 dram of the tincture to 4 ounces of water. Alcohol, bay rum, glycerin and other desiccants which are useful in seborrhea are harmful in senile alopecia. The fat or oil should be used only after the scalp is thoroughly dried. It may be mentioned here that when hair oils, which consisted of animal fats, were used, alopecia was far less prevalent than now.

Hypertrichosis

Excessive growth of hair or a growth of hair in unusual places is a senile phenomenon which cannot be explained by any theory of senescence. It occurs most frequently upon the upper lip of women, appearing during or soon after the menopause, and in the ears, nose and eyebrows of men. The hair is generally of the same color as the hair of the head and it retains its color long after the earlier hair has turned white. It has no significance and nothing ought to be done to remove the new growth. If epilation is desired, the only effective measure is electrolysis. This may, however, leave unsightly spots. The ordinary depillatories do not destroy the roots and they may produce inflammation and possible ulceration.

Canites

Canites or whitening of the hair is a senile manifestation, although it appears occasionally as early as the fourth decade.

The cause is unknown. Metchnikoff has shown that chromophages invade the hair cylinder and carry off the pigment, but in many cases no such organisms can be found. Cases of sudden whitening of the hair are occasionally reported and these seem to support the theory that the coloring matter can be destroyed through nervous influence. It is, however, still undecided if such influence exists or if such reports are warranted by facts. Canites generally proceeds slowly and it cannot be halted. Nothing has yet been found to take the place of hair dyes.

Changes in the Sudoriparous Glands.

There is generally atrophy of the glands with diminished secretion. Occasionally there is excessive secretion in some localities as in the axillæ, about the genitals and anus, and between the toes, and the secretion may have a fetid ordor. Owing to impaired sense of smell this odor may not be obvious to the patient though it be extremely powerful and disagreeable.

Treatment.—*Amidrosis* or deficiency in the secretion of sweat rarely requires treatment. Complete suppression occurs only in fevers and in nephritis, never as a purely senile condition. If a diaphoretic becomes necessary, pilocarpin should not be employed. *Hyperidrosis*, excessive sweating, is seldom so marked as to require treatment. It can generally be controlled by applying a mixture of equal parts of salicylic acid and zinc stearate or one part of tannoform to four parts talcum. Belladonna ointment will suppress the secretion but it may suppress it completely and permanently and this may be as undesirable as the excessive secretion. If there is intertrigo an ointment of zinc oxide 2 drams to vaseline 6 drams should be used and the parts separated by a piece of oiled silk.

Bromidrosis, fetid sweat should be treated like hyperidrosis. The surface should be washed with a 1 to 30 solution of formaldehyde before applying the ointment or dusting powder.

SENILE MUSCULAR DEGENERATION

While progressive atonicity and waste of muscular fiber is part of the normal process of involution we find occasionly rapid or early muscle waste and we may get a pronounced atonicity proceeding to complete paralysis. We may find such paralysis

due to lessened irritability of the muscle fiber without impairment of the nerve supply. In muscles that are not much employed fatty granules deposit in the connective tissue spaces and this fatty infiltration keeps pace with the waste of muscle fibers. Fatty degeneration is a pathological process due to some disease. The pathological progressive muscular atrophy of the young is extremely rare in old age. We find as normal manifestations of senile degeneration of muscle, simple atrophy, atrophy with fatty infiltration and functional impairment, diminished strength, slowed and weakened reaction and lessened electrical reaction.

We sometimes find that the functional impairment is greater than the atrophy would account for and in these cases there is generally fatty infiltration and diminution of nervous irritability. This if excessive, forms a distinct disease.

Progressive muscular enfeeblement of the aged is, according to Oppenheim, due to a multiple neuritis, while Vulpian and Donand have shown that there is always a fatty degeneration of the muscle when this condition exists.

Etiology.—There is, in addition to the normal enfeeblement due to senile waste, some depressive mental or physical influence present. It may be the mental depression caused by the realization of ageing or it may be shock, fear, prolonged pain, a grave disease, neurasthenia, nerve degeneration or insufficient nutrition.

Pathology.—The muscle is pale, soft, flabby and has a greasy feel. The fibers are atrophied, their elasticity is diminished and their striations barely distinguishable. There is a deposit of fat granules between the fibers. No nerve lesions have been demonstrated.

Symptoms.—There is a general progressive weakness, not localized, proceeding more rapidly than the age and the anatomical changes would justify. Slight exertion causes fatigue from which recuperation is slow and not complete. As the disease progresses the fatigue becomes permanent, and any exertion becomes irksome, finally the patient is too weak to arise from bed. There is the will but not the power to move, therein differing from neurasthenia in which the will is impaired but under some stimulus, the patient will move and act as powerfully as in health.

While the disease is strictly confined to the muscles, the lack of activity causes involvement of other tissues. Owing to lessened activity and the consequent lessened waste, less food is required and the diminished metabolic activity causes less heat formation. The temperature is consequently lower, the circulation is slower, aeration proceeds more slowly, gastric digestion is retarded and there is less elimination of waste. Thus various organs and tissues become impaired through lack of activity and fatty infiltration results in them. When the disease has progressed to such extent that the patient will not get out of bed, there is danger of hypostatic congestion followed by pulmonary edema, of bed sores and infection, constipation with autointoxication or of retention of urine. The feebleness may proceed to fatal exhaustion.

Treatment.—The stimulation of muscle irritability is the primary indication. This can sometimes be done by massage, coarse vibration or the galvanic current. Where the cause was mental depression psychic measures may avail. In some cases persuasion, suggestion or harsh measures like threats, a sudden scare or disagreeable medication will be effectual. Harsh measures should never be employed except as a last resort and then only when the physician is certain that the weakness is not due to excessive waste or to some pathological condition of the structure of the muscle. If drug treatment is required we must first determine the condition of the muscles and nerves. Where there is marked fatty infiltration, the iodides are required. They favor destructive metabolism and if combined with passive exercise they will bring about waste of fat. At the same time strychnine, phosphorus, small quantities of alcohol and other nerve stimulants should be used and if the hemoglobin percentage is reduced, hemoglobin should be added.

Localized muscle weakness if not due to traumatism is almost always due to degeneration of the nerve supplying the part.

SENILE ARTHROSCLEROSIS

This disease, first described under the term *senile rheumatism*, is a hardening and stiffening of the joints due to the senile changes in the tissues forming the joint.

Pathology.—The tendons are hardened, there is ossification

Arthrosclerosis of ankles due to ossification of Tendo Achilles and hardening of lateral ligaments. (Courtesy of S. Epstein, M. D., New York.)

and sometimes calcification of cartilages, the cartilages covering the articular surfaces waste through attrition and fibrillation, the ligaments harden and shorten and the synovial sacs become dry.

Symptoms.—The joint gradually becomes stiff but there is never complete anchylosis. The joint is not swollen, reddened or inflamed. There is no pain when it is at rest but there is an ache upon motion, the pain and stiffness increasing with increasing or prolonged motion. Sudden severe motion produces a severe pain. As the patient becomes weaker motion becomes more irksome owing to the increasing stiffness of the joints.

Diagnosis.—Arthrosclerosis senilis is frequently mistaken for chronic rheumatism. There is no history of rheumatic fever, the pains do not get worse at night, there is no pain when the body is at rest and there are no paroxysmal attacks of pain. The pain and stiffness of chronic rheumatism is lessened upon motion or upon "limbering up," as the patient calls it.

Owing to the difficulty in arising from a sitting position, this disease is often diagnosed as lumbago or myalgia. In the senile condition there is hardening and stiffening of the vertebral joints with waste of the muscles of the back, but no myositis. The ache becomes progressively more severe but there is no pain when the patient is at rest or turns in bed. In myalgia the pain comes on suddenly, is generally severe, at times paroxysmal with painless remissions, while in lumbar myalgia, turning in bed is painful.

Treatment.—While the salicylates and local counterirritants are of benefit in the rheumatic conditions they are absolutely useless in the senile condition. The treatment in these cases is purely empirical; in some cases psychic methods in some hydrotherapy, in some drug treatment, is effective. Hot baths or hot applications over the joints followed by inunction with an animal oil or fat is often beneficial. In some cases the best results are obtained from mild or coarse massage or vibratory treatment. The internal medication must be directed to overcome the senile debility, phosphorus, arsenic, strychnine, hemoglobin, etc. Occasionally psychic influences are more effective than drugs and among such influences must be included the empirical use of liniments which the patients themselves employ. The local use of alcohol is contraindicated.

PSEUDO-PAGET'S DISEASE

This name is a misnomer, the disease being a rare form of osteoporosis occurring in old age. The head is held forward, the knees are slightly flexed, and the legs are spread apart. The hands are held out from the body, there are deformities in the lower limbs and trunk, the angle of Louis in the sternum is prominent, and the whole thorax seems to be pushed down into the abdomen. The latter shows a rounded eminence in the epigastrium and hypogastrium, deep transverse folds above the umbilicus ending laterally in depressions in the hypochondriac area, and a diminution or obliteration of the space between the ribs and the crest of the ilium. The spinal column shows one curve with the convexity posteriorly. When the heels are held together the internal condyles of the femurs are far apart. The general appearance is that of Paget's disease but it occurs much later in life, generally in the eighth decade. It is apparently an extreme type of senile degeneration of the bones and a disease to the extent of producing discomfort and deformities. There is no treatment, but braces and a cane can retard the changes and prevent the stoop.

SENILE DEGENERATION OF THE BRAIN

Senile Dementia

The ordinary senile changes in the brain have been described in the chapters on Anatomy and Physiology. In many cases more profound histological and physiological changes occur and give rise to symptoms for the relief of which medical care is indicated. We must remember, however, that there is but little relationship between the organic and the functional changes, that mentality depends upon some unknown quality of the brain cells and not upon size of brain or amount of brain substance, that brain substance has been lost without alteration of mentality or sensory-motor impairment. Memory is most active during the period of development, while reason and judgment increase for years after the brain has reached the limit of growth and even while it is in the process of atrophy. In some the comparative and constructive faculties remain unimpaired to the end of life while the conservative fa-

Pseudo-paget's Disease. Nouvelle Iconographie de la Salpêtrière, Jan.–Feb., 1905.

culty shows diminished power even before the completion of development.

Etiology.—The diminution in the weight of the brain begins normally about the end of the fourth decade of life, soon after it has reached its greatest weight and years before the atrophy could reasonably be charged to diminished nutrition from cerebral arteriosclerosis. The causes that prevail in old age, such as impaired nutrition and neurophages, do not prevail when the atrophic changes begin and no theory has been advanced to explain it. As most of the psychic and somatic functions increase for a time after the brain begins to atrophy, it is reasonable to assume that the waste begins in those cells controlling the functions that are lessened about this time. In what portion of the brain these cells are located is unknown.

We find that organs and tissues which have been insufficiently employed and those which have been used excessively break down early. Activity demands increased blood supply to repair waste, and inactivity lessens the circulation in the part. Prolonged inactivity causes a lessened supply of blood and a slowed circulation with the inevitable result of deficient nutrition and waste of tissue. Excessive activity hastens degeneration partly through fatigue toxins, partly through incomplete repair. There may be also a hastening in the evolution of tissue cells, causing a more rapid development of the cells of an advanced evolutionary stage.

The same causes may apply to the brain cells. Persons of low mentality, such as the uneducated peasants, find it almost impossible to learn anything new after their thirtieth or thirty-fifth year. They may retain the memory of events that have made a powerful impression upon them but they cannot learn a new language, though spending years among those who speak that language. In these persons the cells involved in memory become functionless from disuse and if the same process goes on in them that goes on in functionless cells of other tissues they waste. If on the other hand the conservative faculty has been excessively employed, a rapid deterioration will occur when the functional capacity of the cells has been exceeded. As the mentality differs in different individuals, the functional capacity differs. One child will get brain fag from studying the same lesson that another child of the same age acquires without

difficulty. The enormous amount of information acquired by the child can be gleaned by comparing the school curriculums, age for age, of today with the curriculums of forty years ago. The increase represents only the additional information acquired since that time. The result is that the conservative faculty is excessively employed and by the time that brain development is complete and other faculties are fully active, this faculty begins to become impaired and it is probable that the cells engaged begin to degenerate.

When the circulatory changes of advancing age are active and the nutrition of the brain is impaired, the cell degeneration proceeds more rapidly, reason and judgment become impaired, and at the same time the somatic functions become weakened. Metchnikoff has shown the influence of neurophages upon brain cells but it is not at all certain that these phagocytes appear in every senile case. When they are present they are a potent factor in causing tissue waste.

Pathology.—There are marked changes in the brain tissue and in the vessels. The changes in the latter have been described under Arteriosclerosis. The extent of the arterial degeneration varies, in some cases only the small vessels and capillaries being involved, in others the large vessels alone or the whole arterial system of the brain is affected. Miliary aneurysms are frequent. The changes are fairly uniform in character but differ in degree. When they are far advanced the weight of the brain may be 200 grams below the weight of maturity, the waste being principally in the frontal lobes, the cerebrospinal fluid is increased, the pia mater is thickened over the entire cortex, contains amyloid bodies and plaques of calcareous matter, the dura adheres to the bone and there may be a pachymeningitis interna. The convolutions may be edematous, the sulci are shallow, gaping and filled with adventitious pia. Minute hemorrhages are often found in the cortex and basal ganglion which form the foci for softening. The spongy sieve-like appearance described as Etat-Crible is present. The ventricles are dilated and the ependymal wall is thickened. There is a hypertrophy of the neuroglia, and atrophy of the cortical neurones. The cell bodies become shrunken and diminished in number and the processes become tortuous, narrow and short. The physiological increase in yellow pigment in the cells may proceed to pigmentary degeneration. The changes here de-

scribed are present in advanced cerebral degeneration such as is usually found in senile dementia.

Symptoms.—The functional changes in the senile brain include mental deterioration and physical impairment. In determining the extent of senile impairment, the normal mentality of the individual should be known. The impairment, though manifested in many directions, may progress for years before it becomes obvious to friends and the family who constantly surround him. An early symptom of the deterioration is a hesitancy in recalling names, dates and events, fabricating others if the patient thinks the fabrication will not be discovered. The fictitious name, date or event will probably be forgotten the next day and if the right answer is not recalled the new name, date or event will also be forgotten and another substituted. The patient will forget where he puts things, will repeat questions that had just been answered, forget the names of persons to whom he had just been introduced, and if interrupted while speaking he will forget where he left off or he may forget the subject altogether. Attention is defective and prolonged effort to maintain attention leads to brain fag. The aged person who falls asleep during the play or sermon does so through excessive attention with consequent brain fag and not through inattention or indifference. He must make a sensible effort to understand the connection between things where such connections ought to be instantly obvious. He becomes careless about details and loses the sense of neatness, leaving his desk disordered his room untidy, his clothes disarranged. Business and social affairs are not clearly comprehended and this gives rise to errors. Errors in playing cards, usually charged to inattention, errors in calculation, charged to carelessness, slips of the tongue, absent-mindedness, etc., are due to inability to concentrate attention to one object. He becomes egotistic, exaggerates his own importance and his interests, becomes sensitive to what he considers to be neglect of himself or his interests and thus dislike, hatred, fear and finally oikiomania are developed. This may proceed to delusions of persecution.

There is generally a pronounced change in temperament and emotional attitude. There is frequently depression due either to the recognition of waning powers and the nearness of the closing period of life, or to fancied neglect. The patient

becomes peevish and irritable and will exhibit anger upon the slightest provocation. Elation is rare. His self-interest destroys interest in others and while he may show external sympathy by weeping there is rarely profound grief or joy and these are soon forgotten. The aged take frequent naps during the day, as mental and physical fatigue set in quickly upon activity, and at night they are often restless and walk about aimlessly or they will repeatedly try doors and windows, search through closets, desks, trunks, etc., without any object. Inability to accommodate himself to a progressive order of things is a frequent accompaniment of old age. While there is general mental decadence, in some cases the reasoning faculties are not impaired and where the mental efforts are all directed into one channel, remarkable work may be done in this one direction, but in other directions the impairment is more pronounced. What are usually looked upon as whims, hobbies and peculiarities of great men of advanced age are really manifestations of mental impairment. A deeper grade of mental impairment often seen in the senile climacteric is senile confusion. This condition is marked by disorientation. The patient walks aimlessly and unconsciously, oblivious of his surroundings and often in dangerous localities; and if he can be roused, mental clearness is awakened but almost immeditely dissipated. He loses track of time, distance and direction, goes out in winter without a coat, may undress in the street, speak to strangers, especially to children, talking senseless drivel. Memory may be so weakened that he forgets his own name and if spoken to he shows by his answers that he cannot comprehend or comprehends but imperfectly the import of the question. He is garrulous but there is incoherence of thought with gradually deepening impairment of comprehension until the condition of complete dementia is reached. In this condition the individual resembles either the absolute idiot who had never had a trace of intelligence and is consequently quiet, or—as in terminal dementia—is moving automatically, mumbling, with occasional outbursts of silly laughter, shrieks, etc.

Senile delirium occurs occasionally during the senile climacteric. It is marked by hallucinations and great activity. There are usually the symptoms of senile confusion with short attacks of delirium during which there are delusions and hallu-

cinations that are forgotten when the remission occurs. In some cases the attacks come on suddenly; the patient sitting or walking quietly begins to shout or sing, walks rapidly up and down the room, seeing things or hearing strange sounds. In other cases the attack comes on gradually with restlessness and increasing activity. Prostration may occur, but usually the attack subsides gradually until the patient is quiet. On rare occasions the mind clears up for a short time.

Senile dementia in its medicolegal relations will be treated under that chapter.

The somatic derangements due to cerebral degeneration have not been clearly defined. Impaired coordination is generally of cerebral origin and senile tremor, the increasing physical weakness, paraplegia and diverse impairments of the senses and sensation may be due wholly or partly to cerebral degeneration. Impairment of sensation and of the special senses is often due to a weakened mind, which does not readily interpret the impressions received. We sometimes find that under an extraordinary mental stimulus, as the fear of death in a burning building, the mind clears up, the senses become normal and even strength may be temporarily restored. In such cases the effort is but momentary and is followed by profound reaction.

Treatment.—True senile dementia is progressive and incurable, but much can be done in the early stages of mental deterioration to impede its progress, and in advanced stages it is sometimes possible to temporarily rouse the individual to a comprehension of his surroundings and his condition.

The keynote of treatment is *mental stimulation*. This is opposed to the usual treatment of this condition by rest and quiet. Unless it is a terminal dementia which requires the constant presence of an attendant and mentality is so far gone that no impression can be made upon it, the patient should *not* be placed among insane patients nor immured in an asylum. Our object should be to rouse the patient to take an interest in something else than in his body and his fate. His mental faculties should be constantly employed until brain fag sets in, but mental confusion should be avoided. To give a homely illustration, he may watch a one-ring circus until after a time brain fag will set in and he will fall asleep. If he tries to watch a

three-ring circus there will be mental confusion, possibly delusions, and insomnia. Pleasurable sensations should be produced, especially such as the patient is familiar with. An old popular song will often rouse an aged person out of lethargy, and this is one of the most effectual means to bring about a temporary clearing of the intellect. The concert, ballet or whatever else will produce harmony of color, sound or motion will be beneficial. The monotonous routine of the asylum hastens dulling of the intellect and the association with insane will rouse delusions. As the deterioration increases a constant change of environment, of sight, scenes and sounds is necessary. If there is mental confusion it will be necessary to place the patient in a position where the attention can be concentrated upon one object alone, a view of the sea or distant mountains, a familiar song, or poem, a favorite child or grandchild, etc., changing the object from time to time but always selecting an object pleasing to the patient. In the apathetic and melancholic forms of dementia it may be advisable occasionally to subject the patient to excitement such as a lively seaside resort a masque ball or carnival and though this will produce mental confusion it will stimulate mental activity. The most powerful psychic impressions are often produced by the flattery of young persons of the opposite sex and there is probably nothing that will so effectually produce mental and physical rejuvenescence as a young husband or wife. A recrudescence of sexual desire in the aged without psychic causes, as pornographs, erotic literature, conversation or suggestion, is a symptom of senile dementia. When, however, such desire can be stimulated by psychic measures it indicates a state of mind susceptible to improvement. We can thus explain the remarkable mental and physical improvement in aged men who marry young women.

Of drugs, phosphorus and morphine are the best mental stimulants. Morphine in small doses produces a mental stimulation which passes off in a few minutes or hours. Habituation is, however, rapidly induced and if frequently repeated the stimulating effect is diminished and finally lost unless the dose is constantly increased. If this is persisted in, death will result from morphine poisoning. Morphine in 1/20-grain dose may be used occasionally if temporary mental stimulation is desired. Small doses of cannabis indica will stimulate the imagination,

generally, however, in the direction of delusions and illusions. Alcohol is worse than useless. Phosphorus is the best mental and nervous stimulant we have as it is positive in action and there is no habituation. It should be given when the first symptoms of mental deterioration appear, discontinued when there is a response in a brighter mental attitude and resumed when this passes away. The dose of the ordinary phosphorus is 1/100 grain gradually increased to 1/20 grain, always in solution. For several years the author has used amorphous (red) phosphorus in doses of 1 grain gradually increasing to 5 grains three times a day.

SENILE DEGENERATION OF THE CORD

Senile degeneration of the cord gives rise to numerous functional disturbances. It is, however, often impossible to determine the relation between the structural changes and the functional impairment, as similar histological changes may present the most diverse symptoms, while similar symptoms may be associated with different forms of degeneration or in different locations. Sometimes profound functional impairment exists yet no degenerative change is found after death and extensive areas of degeneration have been found upon autopsy which gave no symptoms during life. Extensive atrophy of the cells of the anterior horns has been found in cases which gave no symptom of progressive muscular atrophy and scattered areas of sclerosis are often found upon autopsy of very old persons, who did not present the symptoms of multiple sclerosis during life. In some cases degenerative changes will be found without symptoms but with a history of earlier nervous disease which has been cured.

The typical senile degeneration of the cord is seldom found before the seventh decade, but the early manifestations of spinal impairment, weakened and slowed impulses and tardy response to impulses, are observed in the sixth decade. Diminished irritability and impairment of the muscle sense are noted about the same time, weakened coordination and lessened muscular power appear later and still later senile tremor makes its appearance. There is an increasing weakness in the lower limbs which in extreme cases may become a paraplegia, but

senile paraplegia is never complete. In some cases the location of the degeneration and the functional impairment do not correspond and we may have a degeneration of the upper part of the cord with no other symptom than paraplegia. In most cases, however, the impairment corresponds with the degeneration of that part of the cord which supplied the impaired tissue. The progressive weakness of the aged is caused partly by the waste of muscle and partly by cord and nerve weakness, and exaggerated by the mental condition of the individual. (The weakness of the cord which is due to its degeneration will be described in the following chapter under the head *Senile Degenerative Myelitis.*)

SENILE DEGENERATIVE MYELITIS

This term is applied to senile degeneration of the cord when its manifestations are pronounced enough to produce distressing symptoms or profound functional impairment. It is not an inflammatory process.

Pathology.—There is usually atrophy of the ganglionic cells, with increase of cerebrospinal fluid, the cord as a whole is firm but there may be spots or patches of sclerosed tissue and occasionally areas of softening are found. The motor areas are usually first affected.

Symptoms.—There is no uniformity in the symptoms or regularity in the order of their appearance. The one symptom present in all cases is a gradual weakening of the lower limbs, the so-called *senile paraplegia.* If the cervical portion of the cord is affected there is motor paraplegia, degeneration of the dorsal portion produces a spastic paraplegia, and if situated in the lumbar portion there is motor and sensory paraplegia. In degeneration of the lumbar portion the reflexes are diminished; if there is dorsal degeneration, they are increased. In many cases areas of degeneration are scattered throughout the entire cord and we may then find exaggerated or diminished reflexes, even an increased knee reflex while the tendon Achilles reflex is diminished or *vice versa.* The paraplegia comes on slowly, is progressive and never becomes complete. The sphincters are rarely involved except when due to local causes. Senile paraplegia is generally associated with *senile abasia* and sometimes with *senile tremor.*

Senile abasia is the slow unsteady gait of the aged when walking without a cane and is due to lessened coordination, muscular weakness, bent knees and the fear of falling. This fear is aroused through the greater difficulty in maintaining equilibrium which now requires a conscious effort. Occasionally there is a slow, tremulous tripping gait, the "abasia trepidante" of Petrens, and when this is associated with senile tremor and senile paraplegia we get a clinical picture resembling paralysis agitans. (For diagnosis see Senile Tremor.)

If the paraplegia is of cerebral origin there is instability on turning around. There is also mental impairment more pronounced than the usual impairment at the patient's age. A myopathic form of senile paraplegia originates in the nerves of the muscles and produces cramps and contractions and later on atrophy. In the spinal form there is no atrophy except the usual waste due to age and disuse.

The treatment of senile abasia is mainly psychic. It is necessary to overcome the fear of falling and to do this we must improve the sense of well-being of the individual. A cane is necessary and it should be so long that the patient need not bend over to grasp the handle when the point on the ground is at the distance of an ordinary step from the body. The medicinal treatment is the same as has been given under senile cachexia. Rubber-heeled shoes should be used and ankle supports and arch supporters should be fixed in the shoes even if local conditions do not make them necessary. The sense of security derived from their use will often overcome the fear of falling and will enable the patient to make a firmer step.

A form of senile paraplegia which Demange calls "*Contracture tabetique progressive des atheromateux,*" is characterized by a progressive contraction of the muscles of the lower extremities with increased tendon reflex. He ascribes the disease to a degeneration of the lateral fibers following atheroma of the smaller branches of the spinal artery. The symptoms have been found without the anatomical changes and the anatomical changes have been found without the symptoms. Sensory impairment is frequently found in the aged but it is often impossible to say if it is due to the brain, cord, nerves or end organs.

Treatment.—As it is generally impossible to localize senile degenerations of the cord, treatment must be applied through-

out its length. The most effective method of stimulating spinal activity is by the application of the faradic current after moist heat had been applied. It is impossible to restore degenerate tissue but it is often possible to stimulate functional activity where it is lessened and that is, after all, the aim of the physician in treating senile cases.

Internal medication is limited to phosphorus, arsenic and strychnine. If there is a luetic taint the iodide of arsenic should be used. The precautions concerning these drugs, given in the treatment of senile cachexia, must be observed. Strychnine is the most powerful of the spinal stimulants, but it must be given in constantly increasing doses and we have no means of overcoming its excessive stimulation of the heart. That organ must be watched when giving strychnine. Palpitation or increased blood pressure is the indication to stop its use. Cold sponges are harmful. Occasionally some intense psychic impression will temporarily restore the power and sensibility of the limbs.

SENILE TREMOR

This is a neurosis for which no pathological lesion has been found, nor is there much known concerning its etiology. Pic says it is a manifestation of an irritable enfeeblement of the nervous system of the neuropath. In most cases there is a neurotic taint, occasionally hereditary; more often there is a history of antecedent nervous disease. (It is placed in the first group on the assumption that it is a manifestation of general debility and a symptom of advanced senile degeneration of the cerebrospinal system.) Some cases are traceable to suggestion or mimicry.

Symptoms.—Senile tremor generally begins with an unsteadiness of the hand which has done the most work, then both hands are affected and slight muscular exertion causes them to tremble. Later the head and neck muscles are involved. The lower limbs are rarely affected except after considerable exercise, such as a long walk, taking long steps, climbing stairs or swinging the legs when bent at the knee. It is a slow intention tremor, the oscillations not exceeding five per second, and coming on only during the voluntary contraction of the muscles. The tremor is most noticeable in the head and neck muscles,

there being a coarse tremor of the head, a fine tremor of the lips, and a shaking or trembling of the lower jaw. The shaking of the head is generally in an up and down (vertical) direction but a tremor in a horizontal direction and even a rotary motion have been observed. The tremor of the lower jaw resembles the motion of mumbling, occasionally that of chewing.

The tremor is increased upon excitement or exertion but may be temporarily controlled by the will. In some cases the tremor is confined to the hands during voluntary motion.

The head does not tremble when supported by the hand and the tremor ceases during sleep.

Diagnosis.—Senile tremor is diagnosed from paralysis agitans by the absence of the characteristic attitude, gait, muscle stiffness, cramps, forward pitch, posture of the hands and the immobile facial expression of the latter disease. If senile tremor is associated with abasia trepidante, there may be a similar gait and a tendency to pitch forward, but the other symptoms of paralysis agitans are absent. The tremor of the hands in senile tremor resembles the tremor of disseminated sclerosis but the latter is a disease of early and middle life, and is characterized by a spastic gait, a rapidly increasing weakness, nystagmus with other ocular symptoms, and peculiar scanning speech, all of which are absent in senile tremor. The toxic tremors, as from alcohol, tobacco, lead, mercury, etc., are intention tremors but the history and accompanying symptoms readily distinguish them from senile tremor. In hysterical tremor other symptoms of hysteria and the absence of tremor when the attention is diverted, determine the diagnosis.

Treatment.—Drugs useful in other tremors have no effect upon senile tremor. Neither hyoscine, hyoscyamine, duboisine, the bromides nor iodides are of any use. The only drugs which seem to have any effect are arsenic and strychnine in gradually increased doses to the limit of tolerance of the former and until palpitation of the heart indicates that the strychinne is acting unfavorably upon that organ. Psychic measures, especially flattery, will sometimes cause arrest of the tremors by concentrating attention to them and rousing a persistent effort to control them. If the tremor is due to imitation, harsh measures, such as threats, deprivation of food, etc., may be necessary to effect a cure.

SENILE DEGENERATION OF THE NERVES AND END ORGANS

Pathology.—The degenerative changes in the nerves are a hyperplasia of the neuroglia and an atrophy of the nerve cells and fibrils, more marked in the terminal fibers and diminishing toward the center. The senile changes in the nerves appear late in life, indeed in many cases no histological changes can be found to account for symptoms evidently due to the nerves. It is often impossible to say whether a neuralgia, local paralysis, motor or sensory impairment, tremor or reflex perversion is due to cerebral, spinal, nerve or end organ defect. In some cases giving the symptoms of neuritis we can find the pathological changes observed in chronic interstitial, parenchymatous or multiple neuritis but in most of these cases we can discover an etiological factor besides senile involution.

Symptoms.—The functional activity of the nerves is diminished, motor impulses are slowed and weakened, sensibility is lessened, the special senses are impaired and the functions of the regulation centers are slowed, weakened and sometimes perverted. The loss of muscular strength is due partly to the nerve changes and partly to waste of muscle and lessened muscle irritability. The impairment of the special senses may be due to degeneration of the afferent nerves or it may be due to some change in the end organs or in the brain. It is probable that the nerves and end organs are both involved while the brain is less able to receive and interpret sensory impressions. Fear exaggerates the sensation of pain and the aged complain of acute pains where the local condition does not give rise to much pain. The patient may complain of intense pain from a scratch which he can see, yet he will hardly notice the pain from a peritonitis, pleurisy or acute gout when he cannot see the diseased tissue. Suggestion and mimicry will give rise to painful sensations without any lesion of the part or of the nerve supplying it. It is important in examining a patient for painful spots that no hint be given of the object of the examination, lest the patient should declare that any spot is painful over which greater pressure is made or to which his attention is directed.

Little can be said of the regulation centers. The heat regulation, cardiac regulation, vasomotor centers, etc., are all

impaired but they have not been sufficiently studied to make definite statements about them.

The *optic nerve* and retina are rarely affected but there may be a degenerative albuminuric retinitis associated with a generalized arteriosclerosis and chronic interstitial nephritis. Optic neuritis or choked disc is occasionally found under the same circumstances. The *motor nerves of the eyeball* show neither histological nor functional changes. There is often a slowness of action of the motor muscles which may be due to muscle weakness or to slowed mental impulse or response. The terminal ends of the third branch of the *trifacial nerve* are sometimes subjected to pressure in the bony structure of the lower maxilla and this gives rise to trifacial neuralgia (see Trifacial Neuralgia). It is supposed that neuralgia of the second branch is due to arteriosclerosis and consequent impaired nutrition, although this form of neuralgia often appears with no demonstrable lesion in either nerve or blood-vessels. The lingual branch shows frequently functional impairment in diminished taste without histological change.

The *facial nerve* is rarely if ever affected as the direct result of senile changes. When facial paralysis occurs, it either follows cerebral disease, or a neuritis, or it is due to exposure, traumatism, pressure or other cause not connected with the senile processes.

The *glossopharyngeal nerve* shows in the aged frequent functional impairment in diminished taste. It is, however, uncertain to what extent degeneration of the taste buds contribute to this result. The dysphagia of the aged is due to lessened innervation of the muscles of deglutition. Various functional perversions in parts supplied by the *vagus* are believed to be due to senile degeneration of that nerve. To this cause are ascribed the anomalies in the rhythm of the heart and of respiration, pharyngeal and laryngeal spasm, aphonia, and most gastric neuroses.

The *spinal accessory nerve* shows no marked functional change in old age. It is probable that the weakening of the sternomastoid and trapezii is due to weakened power of this nerve, but the weakness of these muscles is rarely more marked than in other voluntary muscles.

The *hypoglossal nerve* presents no symptoms that can be ascribed to senile degeneration.

Senile Degeneration of the Organs of Special Sense

The special senses are weakened or perverted in old age but we can rarely tell with any degree of certainty whether the fault lies in the terminals, nerves or cerebral centers, nor whether the cause is senile involution or something else. In many cases no histological changes can be found.

Anosmia, loss of smell, may be due to obstruction of the passage of air to the Schneiderian membrane, to atrophy or degeneration of this membrane or of the olfactory nerve or bulb, or there may be a senile dementia with diminished power to receive or interpret sensory impressions, or it may follow other nasal diseases or hysteria. The loss of smell may be confined to certain odors or it may be more marked at certain times, as in damp weather. The sense of smell is usually the first of the special senses to show diminution of power, the impairment, however, is rarely noticed and complete anosmia may exist without the knowledge of the individual.

Parosmia or a perverted sense of smell may be due to the same causes as anosmia, the perversion preceding the latter, or it may be due to mental aberration, hysteria or neurasthenia or to oral or nasal disease producing a fetid odor. It is often an early symptom of insanity.

If the anosmia is due to senile degeneration nothing can be done to increase the sense of smell. The treatment of parosmia is either local or psychic depending upon the cause.

Gustatory anesthesia generally accompanies anosmia but it rarely proceeds to the same extent. Taste may be lost for certain substances, or only the temperature sense may be affected. There is usually a blunting of the sense of taste for alkaline, sweet, insipid and bitter substances but not for salty, acid or acrid ones. The impairment may be due to a change in the taste corpuscles, in the nerves of taste or in the cerebral centers. Probably all three are responsible for the functional impairment, the papillæ being mainly affected through the changes in the covering membrane of the mouth and the changes in the oral secretions.

Gustatory paresthesia is rare except in insanity or hysteria. Nothing can be done to increase the sense of taste due to senile degeneration.

Presbyacusia, diminished sense of hearing, due to age is frequent and often proceeds to complete deafness. The appreciation of high notes is generally lost and low notes appear higher. Tinnitus and other abnormal sounds indicate a pathological condition; they do not appear in the ordinary senile impairment of hearing. They are generally associated with cerebral or local arteriosclerosis. Presbyacusia occurs earlier in the cities than in the country and when found in city dwellers it proceeds more rapidly and is more frequently subject to complications which convert the normal process into a disease. The character of the senile process is unknown as both atrophy and thickening of the drum have been found in the aged, with various degrees of deafness and without functional impairment, while complete deafness may exist without drum or nerve change, or any other known cause. It is believed that many cases of senile deafness are of cerebral origin.

Presbyacusia is sometimes overcome by the use of a speaking tube, mechanical drum or other appliance which will cause more direct conduction or intensify the sound. Drugs and operative procedure are useless.

Presbyopia or difficulty in accommodation to near objects is the ordinary condition of the senile eye and is due to sclerosis of the lens with probable weakening of the muscles of accommodation. The contracted pupils and slowed reflexes are probably due to changes in the nervous supply and in the muscles. No distinctively senile changes have been found in the retina or optic nerve and in those who have not abused their eyes the sense of sight remains normally acute. Presbyopia like presbyacusia occurs earlier in the cities, it proceeds faster and complications are more frequent. Amblyopia is frequently found in the aged; it is, however, not a senile condition. The treatment for presbyopia is appropriate glasses. Perversions of sight and hearing that are not insane illusions may occur in cerebral and auditory arteriosclerosis but they do not occur in the ordinary senile degenerations of the organs.

Anesthesia is frequently met with in the aged. It is usually partial, there being a weakened perception of touch, pain and

temperature. Complete loss of sensibility occurs only in some forms of spinal degeneration and is then associated with motor paralysis. No senile anatomical change of a degenerative character has been demonstrated in the tactile organs and it is uncertain whether the functional impairment is due to slowed conduction, weakened cerebration or to some morphological change which has hitherto defied recognition.

Hyperesthesia is rare in old age, and the excessive sensitiveness from which the aged often complain is generally exaggerated through the fear of pain.

Paresthesia occurs frequently in the form of pruritus and occasionally as formication.

Senile Pruritus

Etiology.—This is one of the most frequent and most annoying of the ailments of old age. It is sometimes due to psychic causes, either suggestion or mimicry. Epidemic senile pruritus in institutions has been traced to mimicry, one patient suffering from a pruritic affection and others seeing that patient scratch, do likewise. The mere mention of an itch-producing cause, such as fleas, will sometimes suffice to arouse the sensation of itching. If we can exclude these psychic causes as well as the pruritic dermatoses presenting surface lesions, parasites, also the diseases which are usually accompanied by pruritus, such as diabetes, jaundice, leukemia, pseudoleukemia and nephritis, and finally all local causes of irritation, such as woolen underwear, acrid bromidrosis, etc, we necessarily deal then with the true senile pruritus of unknown etiology and no discoverable pathology. Lesions may appear, due to scratching, but no change has been found in the end organs to account for the pruritus itself.

Symptoms.—The itching may be protracted, intermittent or ephemeral, so mild as to be barely noticed or so severe as to cause intolerable agony, it may be generalized, scattered over large or small areas or localized. When localized it is generally found about the genitals and anus, sometimes about the legs, rarely in other locations. The itching is usually worse at night, in damp weather and after excitement.

Diagnosis.—In dealing with senile pruritus we must first eliminate other forms of pruritus. This is comparatively easy

when it accompanies internal diseases or when due to pruritic dermatoses presenting surface lesions. There are no lesions in senile pruritus unless excoriation, dermatitis or eczema is produced by scratching. In these cases the irritation long preceded the scratch lesion.

Pediculi and the acarus are the principal dermal parasites and when present are found without difficulty in the locations which they infest.

The thread worm, oxyuris vermicularis, though rare in the aged, may be present and give rise to an intolerable itching about the anus. It is often difficult to determine the cause if the pruritus is due to some local irritant. The most frequent source of such irritation is acid or acrid perspiration, sometimes woolen or flaxen underwear will cause it, occasionally handling irritating substances will produce it. In all cases in which there is a dermatitis or a local hyperemia not due to rubbing we can exclude senile pruritus. It is sometimes impossible to decide when the pruritus is due to psychic causes. In one institution where several sufferers from pruritus charged the original case with spreading phthiriasis among them, it was necessary to isolate all patients and scrub them, although none had pediculi.

Treatment.—There is no specific treatment for senile pruritus. The same measures which are apparently successful in some cases are detrimental in others, while in some cases the itch will suddenly disappear after all treatment had been discontinued. The only drug which can be depended upon to give temporary relief is cocaine in a 2 per cent. ointment, using lanoline or sweet butter as a base. Occasionally a single application will give permanent relief, usually, however, the itch returns in a few hours. In some cases hot water, in some again cold water applications are of service. Ice and freezing mixtures will relieve the itching but may produce frost bites followed by gangrene. Weak acid solutions, alkaline solutions, menthol, thymol, irritants like capsicum and cantharides, sedatives like belladonna, stramonium and chloral, have all been recommended and the faradic current has been employed with benefit. In a disease of this character, with unknown etiology and pathology, with a single distressing symptom, our efforts must be directed to the relief of that symptom and such measures can be used but empirically.

VARICOSE VEINS

Varix is the most frequent affection of the veins in old age and a mild type becomes physiological with advancing years. As the senile changes in the heart and blood-vessels proceed, the venous circulation becomes slower the veins become overfilled and they dilate.

Etiology.—The normal varix of old age is seen in the superficial veins of the hands and feet. When this is excessive or due to other causes than the normal changes in the veins and the slackened venous circulation it is pathological. The most frequent site of this form of varix is about the lower limbs. It is found mostly in women who have had children and is then carried over from maturity. Among men it is generally found in those who stand or walk much. Arteriosclerosis and phlebosclerosis is generally present and it is believed that in almost every case there is an obstruction to the return circulation due to tricuspid stenosis. (Hemorrhoidal varix will be treated under Hemorrhoids.)

Pathology.—The vessel becomes longer and dilatation occurs generally just above the valves. The vein assumes an irregular or wavy line with a single globular enlargement or there may be a series of dilatations giving the vessel a beady appearance. In some cases the dilatation extends in an unbroken line for some distance along the vein. The coats are generally hypertrophied except at the dilated portions which are thin. Calcareous plaques are sometimes found in the walls and thrombi form in the dilated pouches.

Symptoms.—The veins present the familiar dark blue, wavy or irregular appearance seen upon the hands of the aged. Where the varicosity is pronounced the vessel appears swollen at one place, or there may be a string of such swellings, or the swellings may be scattered showing that several vessels are involved. The leg feels heavy and after long standing a hypostatic edema sets in about the ankles.

Varicose veins are subject to many complications. The more important ones are pruritus, eczema, erysipelas, thrombus rupture and ulcers. Eczema is generally the result of scratching or rubbing where there is an intolerable pruritus and the same cause may be followed by rupture, ulcer and erysipelas.

Treatment.—Mild varicosities require no treatment. When pronounced the treatment is either radical by surgical procedure or palliative by means of bandages. There is some danger in rubber bandages, as they may compress the arteries as well as the dilated veins and cause impaired nutrition of the limb, unless they are applied evenly and with just enough tension to compress the varix without compressing the limb. A close fitting rubber stocking is better. For the intolerable itching a 2 per cent. cocaine lotion or ointment can be used. Sometimes ice will give relief, but the usual antipruritic remedies are generally worthless. If the pruritus is relieved, the eczema can usually be cured by the application of stearate of zinc, oxide of zinc or bismuth subnitrate. As long as the pruritus exists no treatment of the eczema will avail as it will be impossible to keep the patient from scratching.

Hemorrhage from rupture is generally controlled without difficulty by compression below the site of rupture. Owing to the slowed circulation a clot will be speedily formed and if care is taken to prevent infection repair will take place. (Erysipelas is treated under the Infectious Diseases and Ulcer under Diseases of the Skin.)

SECONDARY SENILE DISEASES

The diseases of the second group are always secondary although the primary condition may be so obscure as to be unknown or unnoticed before the secondary disease appears. Typical examples of this group are apoplexy, senile gangrene, and angina pectoris.

THROMBOSIS AND EMBOLISM

These occur rather frequently in the aged. Though usually described together they differ so greatly in their etiology, pathology, symptoms and prognosis that they will be considered separately.

THROMBOSIS

Etiology.—Thrombosis may be due to damage to the lining membrane of the vessel, to slowed circulation, or to a change in the character of the blood whereby its coagulability is increased. In old age, all three causes usually prevail, thus ac-

counting for its frequency at that period of life. It may occur in either arteries or veins, the latter being more often affected, the circulation being slower there; especially is this the case in the lower extremities and in the brain. The damage to the vessel is usually due to an arteriosclerosis or fatty degeneration, the site of the lesion being the focus for the deposit of the agglutinated blood cells which form the primary thrombus. Varicose veins are frequently the seat of thrombosis. Other causes less prevalent in the aged are inflammation, toxemia, dilatation of the vessel or of the heart. Some of the infectious diseases cause thrombosis, either by direct effect of the toxemia upon the lining membrane of the vessel or by changing the character of the blood by which its viscosity is increased.

Slowed current may be due to cardiac dilatation or weakness, or to dilatation of the vessels as in varicose veins. The senile changes in the blood are unknown. Its viscosity is increased and its coagulability is consequently greater than in maturity but it is uncertain whether there are other changes also which tend to cause agglutination of the cells and adhesion of the blood plates to the walls of the vessel. Traumatic causes, as compression of a vessel, may produce thrombosis and thrombi are frequently found in the heart, either as small vegetations or as adherent coagula. These are probably formed shortly before death.

Pathology.—Whether occurring in an artery or in a vein the pathological process is the same. In the normal flow the red cells and blood plates keep to the center of the stream while the white cells travel along the wall of the vessel. When the circulation is slowed the plates leave the center and accompany the leucocytes. They will either adhere to the healthy endothelium or, finding a spot which is broken down, this spot becomes then the focus for the thrombus. The fibrin element parting from the plasma, the plates and the fibrin form the primary layer or primary thrombus upon which layer after layer of plates and fibrin are deposited. The caliber of the vessel is diminished and it may be entirely obliterated. This usually proceeds slowly and in many cases collateral circulation is fully established before complete obliteration takes place. When occurring in a terminal artery, the tissue supplied from the vessel beyond the occlusion is deprived of nutrition and in-

farction results. In some cases the thrombic formation proceeds so rapidly that complete occlusion occurs before any compensatory collateral circulation is established and gangrene results. This occurs most frequently in thrombosis following infectious diseases and is rare in the aged. In venous thrombus there is passive congestion and edema of the part below the occlusion. The thrombic deposits are sometimes removed by a process of softening, while sometimes they become organized and form part of the vascular wall. In some cases particles are torn off and are carried in the circulation as emboli.

Symptoms.—The symptoms of thrombosis depend upon the location and rapidity of formation.

The most frequent location of thrombosis in the aged is in one of the cerebral vessels. (The symptoms are described under *Cerebral Softening* while cardiac thrombosis will be described separately.)

Venous thrombus occurs most frequently in the veins of the lower extremities, occasionally in the cerebral sinuses and veins, rarely in other veins. In the aged the most frequent cause of venous thrombus is phlebosclerosis with slowed circulation, rarely a phlebitis. In the lower limbs the usual location is in the dilated portions of a varicose vein. Sometimes the deposits can be felt as hard lumps which cause pain when pressed upon. There is generally pain and edema upon standing, both subsiding when the limb is in a horizontal position. If there is a phlebitis the pain is constant and severe, the edema is extensive, there is fever and other symptoms of inflammation and the thrombosis progresses rapidly. Phlebitis is, however, very rare in old age and when it does occur it is almost always due to traumatism. The principal danger from venous thrombus lies in the detachment of particles which are carried to the heart and then to the lungs as emboli.

Sinus thrombosis is rare in the aged, and when occurring it is almost always secondary to an injury, inflammation or other pathological condition in or about the skull. Primary thrombosis of the longitudinal sinus has been found upon autopsy which gave no symptoms during life and for which no cause could be found. There are no clearly defined symptoms pointing to this disease. There is usually headache, dizziness, mental depression; there may be convulsions, hemiplegia, and other

cerebral symptoms, but all these may be due to the primary disease or may be associated with other conditions. Fulness of the veins of the face and head and local edema are fairly indicative of sinus thrombosis but they are not always present. In many cases the disease is of septic origin and then there will be a jugular phlebitis with symptoms of pyemia.

Treatment.—(The treatment of gangrene, cerebral softening and cardiac thrombus will be given under those heads.) The treatment of sinus thrombus is surgical and must be directed to the causative condition. In the venous thrombosis of the lower extremity, if due to phlebitis, the latter must be treated. Absolute rest, the limb being rendered immobile by splints, is imperative. Local applications of hot water or hot lead water should be made. If there is an underlying septic condition that must be treated, and if these measures fail surgical interference may be necessary. If the thrombus forms in a varicose vein, rest and the application of tincture of iodine or iodide of potassium ointment, with strapping of the limb, may effect a cure.

Cardiac Thrombosis

At almost every autopsy a clot is found in the heart. It is probable that most clots are formed shortly before death when blood changes favor coagulation and the slow current permits their adhesion to the walls of the cavities. In many cases, however, the clots are evidently of long standing, firm and closely adherent to the walls.

Etiology.—The causes of cardiac thrombi are obstruction to the passage of blood through the heart as in valvular defects, slowing of the current through dilatation or atonicity of the myocardium, change in the character of the blood whereby its coagulability is increased as in infectious diseases, nephritis, diabetes, etc., and roughening of the endocardial surface. Small, firmly adherent, slowly developing thrombi, called vegetations, occur frequently after rheumatism and in the various valvular degenerations.

Pathology.—The vegetations are usually fibrinous adhesions to the valves or cordæ tendinæ. They are of the same consistency as the adjoining tissue, lighter in color with irregular

edges. Larger and more recent thrombi may reach the size of a walnut and may be found in any cavity. They may be firm but are usually soft and jelly-like, while in diseases running a rapidly fatal course they are very soft. Older thrombi consist of exsanguinated fibrin, are light in color and are usually closely adherent to the endocardium.

Symptoms.—The vegetations rarely give any pronounced symptoms except where they extend beyond the edges of valves and thus produce stenosis. The symptoms then are the symptoms of valvular stenosis. If a thrombus forms rapidly and is of large size it may cause a speedy fatal end by complete obstruction of the cardiac circulation. If smaller or developing more slowly there is dyspnea, cyanosis, partial syncope, irregular and weak heart action, hurried respiration, restlessness and anxiety, later cerebral symptoms, pulmonary edema and finally coma. In some cases death occurs in a few hours. A small thrombus may give less severe symptoms but the condition is always grave and generally fatal.

Treatment.—There is no known treatment for this condition. The alkaline carbonates are supposed to have the power to prevent coagulation but neither these nor the iodide of potassium relieve the symptoms or prevent death when a large thrombus has formed. In milder cases rest and small doses of digitalis and opium may be tried.

EMBOLISM

Embolism occurs more frequently in the aged than in earlier life, as some of the principal sources of the embolus are mainly found in the aged. It may be, moreover, directly responsible for several senile pathological conditions, such as cerebral softening, senile gangrene, etc.

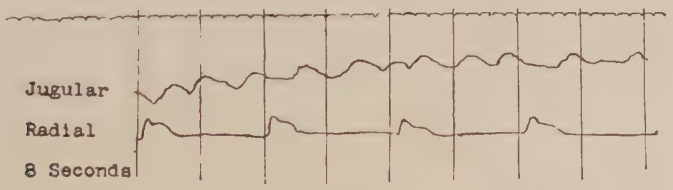
Etiology.—Embolism is a secondary disease, the plug itself being a pathological product of some other disease. It may be a fragment of a blood clot detached from a thrombus, a particle of calcareous or other matter from an atheromatous plate, vegetation from the endocardium, a piece of neoplasm, a mass of bacteria or a mass of pigment. It may be a fat embolus, or hyaline matter or any other substance that has made its way into the circulation. The plug is carried in the circulation, and if coming from a vein or the right side of the heart, it lodges in

the pulmonary artery or in one of the branches; if coming from the left side of the heart or from an arterial source it is carried to some smaller vessel, which it blocks. Thrombic or atheromatous fragments or vegetations may become detached when the heart action is suddenly increased by exercise, drugs or a sthenic inflammation and the blood current is sent through the diseased vessel with greater force.

Pathology.—Plugging of an artery or arteriole causes anemia and degeneration or gangrene of the tissues supplied by the part beyond the plug, unless anastomosis and collateral circulation is speedily established. In embolism of smaller vessels and arterioles, except in terminal arteries, anastomosis generally occurs. If a larger vessel is blocked or when it is a terminal vessel, infarction, degeneration or gangrene follows, or it may cause complete functional arrest and death as when the pulmonary artery, coronary artery or one of the larger cerebral vessels is blocked. In some cases the lumen is not completely plugged by the embolus. Fibrin is then deposited upon it and a thrombus is formed which may completely block the vessel. If this occurs in a larger vessel the part beyond the plug is not immediately cut off from nutrition and collateral circulation may be established before the lumen is closed entirely.

Symptoms.—*Pulmonary embolism* occurs when the pulmonary artery or a branch is blocked. The embolus is either carried from a vien, being a detached portion of a thrombus, or it originates in a vegetation of the right heart. In old age when the venous circulation is normally weak, degeneration of the endocardium of the right heart leads to the formation of a thrombus which may become dislodged by a powerful cardiac contraction and carried to the pulmonary artery or may occlude one of its branches. If a main branch is blocked there will be instant collapse and death. If a small vessel is closed up a hemorrhagic infarction will result. This begins with a sudden, severe pain, dyspnea, a feeling of oppression and anxiety, sometimes a chill. The heart is weak, there is usually cyanosis and blood-streaked expectoration and there may be small blood clots in the sputum. The severity of the symptoms varies with the extent of pulmonary involvement, a minute infarction giving no pronounced symptoms except perhaps the expectoration of small blood clots. Dark scanty hemoptysis is the

Adams-Stokes disease, showing the patient just recovering from a syncopal attack, with rapid pulsation visible in the depression above the clavicle. From "*Heart Disease and Blood Pressure*" by L. F. Bishop, M. D. (Funk & Wagnalls, New York.)



Tracing of the jugular and radial pulses during eight seconds. (From "*Heart Disease and Blood Pressure*" by L. F. Bishop, M. D. (Funk & Wagnalls, New York.)

pathognomonic sign of pulmonary infarction (Loomis). The condition is serious even with a small infarction. Embolism in the brain produces cerebral softening which will be described separately.

Portal embolism may occur from embolic matter brought from the stomach, intestines, spleen or pancreas. The free anastomosis of the portal and hepatic systems whereby rapid collateral circulation is established prevents the formations of infarctions. Portal embolism gives no symptoms unless there is complete occlusion, when the symptoms are the same as in the hepatic obstruction of cirrhosis of the liver.

Renal embolism produces renal hemorrhagic infarction. The embolus generally comes from a fragment of endocardial or valvular vegetation which was torn away by some powerful cardiac contraction as after exercise, or from the loosening of some atheromatous matter. The symptoms are fever, an ache or pain in the the region of kidneys coming on suddenly and persisting, blood in the urine, and symptoms of cardiac disease or arteriosclerosis.

Femoral embolism is rare in old age. When it does occur there is a sudden severe pain, the limb is blanched and numb, followed by complete loss of sensation and motion. If the occlusion is complete and collateral circulation is not rapidly established gangrene sets in, beginning in the foot and extending upward. Partial occlusion or incomplete collateral circulation causing imperfect nutrition of the part beyond, will result in atrophy of the limb with impaired motion and sensation. Embolism of a branch of the femoral or in another vesssel of the lower extremity produces the same result, the extent depending upon the extent of the occlusion, the rapidity and extent of the collateral circulation and the amount of tissue which has been deprived of nutrition.

Air Embolus.—This occurs when air enters a vein. If it is a minute globule it produces a momentary arrhythmia on reaching the heart. A larger amount of air will produce a spasm of the heart and may cause fatal collapse.

Mountain Sickness.—The rapid ascent of high altitudes is liable to produce cardiac thrombosis and pulmonary embolism in aged persons. The attack may occur several days after the descent and is fatal.

Treatment.—A large pulmonary embolus is rapidly fatal. If it is a small embolus causing an infarction, our main reliance is in absolute rest and the treatment of symptoms as they arise. Cardiac stimulants are contraindicated as they may dislodge further emboli. If the circulation is weak, hot water should be applied to the feet, and dry cups and sinapisms to the back and chest. If there is great dyspnea morphine and atropia should be used. When the acute symptoms subside iodide of potassium should be given to promote absorption.

No treatment is required in a portal embolism. If complete occlusion occurs it should be treated the same as cirrhosis of the liver.

In renal embolus, tannic acid in 5-grain doses will control the hemorrhage, but ergot, which is of service in this disease in earlier life, is inadmissible if there is arteriosclerosis. Diuretics are contraindicated. Iodide of potassium may be used in small doses to secure absorption and elimination of the degenerated tissue.

In embolism of the femoral or other artery of the lower extremity, active local hyperemia should be produced and maintained to stimulate collateral circulation. If this fails and gangrene sets in surgical interference will alone avail to save the limb or the patient.

SENILE GANGRENE

Senile or atheromatous gangrene is a dry gangrene due to tissue starvation through an obliterating arteriosclerosis. The dry gangrene following embolism, thrombus, traumatism or chilling of the surface, does not differ in pathology or symptoms from the other and when occurring in the aged is included in the term of senile gangrene. Moist or septic gangrene which is due to infection or to diabetes is not strictly a senile gangrene but will be included here for the sake of continuity of description.

Etiology.—The cause of senile gangrene is the closure or obliteration of a vessel where collateral circulation is not rapidly established. This may occur as the result of an arteriosclerosis or embolus, less frequently a thrombus. It may also occur when the surface has been chilled and the circulation is poor, the vessels contracting and the circulation ceasing in the part involved.

Photograph of Heart in a case of Adams-Stokes disease—showing Calcareous nodules (center of picture just below the aortic valves) in the region of the bundle of His. (From "*Heart Disease and Blood Pressure*" by L. F. Bishop, M D. (Funk & Wagnall's, New York.)

Traumatism may cause gangrene through injury to a vessel or through pressure upon a part, as occurs in bedsores. In this case there may be either destruction of the part through direct compression or through the compression of the nutrient vessels. In many cases of senile gangrene there is a history of gout, rheumatism, syphilis, alcoholism, or infectious disease, all causes for chronic endarteritis with consequent thrombosis or embolism. Some are the terminal stage of Raynaud's disease. Moist gangrene may follow an infectious disease, infection of a surface lesion or some disease like diabetes, nephritis, or cerebral disease which diminishes the resistance to infection.

Senile gangrene occurs most frequently in one of the lower extremities, generally in a foot or toes, occasionally in an upper extremity, rarely in two membranes at the same time or in other parts of the body, except when frozen or injured.

Symptoms.—The earliest symptoms of senile gangrene is a tingling or feeling of numbness in the part, the part becomes pale and cold and later it is livid. If occurring in the foot, the latter feels heavy and cold and its sensibility is diminished. After a time a brownish or purplish spot or patch appears, which increases in extent and becomes darker until it is almost black. The skin over this area is dry, hard, and leathery, and may exfoliate. The area involved is insensible to pressure or punctures, sensation and motion being completely lost. The tissues become mummified. The destruction proceeds until all the tissues in which the blood-supply was cut off are involved. In most cases there is a line of demarkation where the gangrene stops and the part destroyed falls off. In some cases there is no line of demarkation and the destruction of tissue proceeds in all directions. This occurs more frequently in moist gangrene. In this form the tissues become soft and pulpy and putrefy with the formation of pus. There is the odor of decomposition and the tissue resembles the slough of an ulcer. There is usually some pain in the beginning but this soon gives way to tingling numbness and insensibility. In septic gangrene there are the usual symptoms of septicemia, rigors, irregular fever, perspiration, rapid pulse, some stupor and typhoid symptoms.

Senile gangrene due to thrombus or arteriosclerosis proceeds slowly, the paling and tingling being so slight at the beginning as to barely attract attention, gradually becoming more intense.

The gangrene due to embolus generally begins with a sharp pain followed by tingling or numbness, and the disease progresses rapidly. The prognosis is good as to life if a line of demarkation is formed and the area involved is small. Aged persons have recovered after amputation at the hip-joint. As for the part involved the prognosis is unfavorable. It is sometimes possible to arrest the disease with but slight loss of tissue; even complete recovery has been effected. Moist gangrene proceeds rapidly, there is no line of demarkation and it is almost always fatal.

Treatment.—The treatment depends upon the cause and the stage of the disease. Tissue that has already become gangrenous must be removed. If septic symptoms appear rapid excision or amputation is imperative. Temporizing is fatal in such cases. In the early stage of dry gangrene it is sometimes possible to bring about rapid collateral circulation by applying hot water constantly to the part. It should not be raised. In the gangrene following arteriosclerosis, iodide of potassium should be given in 5-grain doses every four hours until the physiological effects of iodism appear, in order to produce lessened viscosity of the blood.

If it is a frozen part heat should be applied gradually and the tissue massaged. When operation becomes necessary, if there is no line of demarkation, amputation must be performed at the joint above the lesion.

CARDIAC NEUROSES

These are functional disorders involving temporary or permanent change in force, frequency or rhythm of the heart. These anomalies are in some instances symptoms of organic disease of the heart itself or of the coronaries, or of gastric or cerebral disturbance, fever, toxemia, pain, etc.; in some cases they are due to non-pathological causes, such as menopause, high altitudes, hot baths, exercise, excitement or exhaustion, the use of tea, coffee, tobacco, etc. They may also be due to senile changes in the nervous regulation of the heart either in the vagus or in the intrinsic ganglia, or to some alteration in the muscular structure. The heart beats should be counted and their character determined at the heart and not

at the radial pulse, as radial arteriosclerosis may alter the character of the latter, or beats may be lost between the heart and wrist, or the ventricular contraction may be so weak that the impulse is not carried with sufficient force to distant vessels to give a palpable pulse. The pulse, moreover, gives no indication of the condition of the auricles.

When these changes in the force, frequency or rhythm of the heart are due to diseases presenting anatomical lesions, as in valvular disease, or are symptoms of clearly defined pathological conditions, such as fever, they cannot be considered as neuroses. It is, moreover, probable that every neurosis is dependent upon some temporary or permanent change in the structure or character of the tissue, the function of which is impaired. So long as we have not determined what that change is, we class such functional disturbance as a neurosis or psychosis.

Palpitation

This is an alteration in the force, frequency or rhythm of the heart which becomes noticeable to the individual.

Etiology.—When permanent, it is a symptom of organic heart disease, exophthalmic goiter, or a continuance of a non-pathological cause, as excessive smoking, tea, coffee, alcohol, sexual indulgence, etc.

Temporary palpitation of the heart may be due to any of the non-pathological causes mentioned, to other neuroses, to upward pressure upon the diaphragm from a distended stomach, irritation of the nervous system, strong emotions, even pleasurable anticipation, or abnormal condition of the blood as in anemia, uremia, or other toxemias. It also occurs in uterine and ovarian disorders. In some cases no cause can be found.

Symptoms.—The pathognomonic symptom is a more or less violent thumping, beating or fluttering of the heart perceptible to the patient. When due to organic heart disease there is generally, arrhythmia with the other symptoms of the underlying condition. In exophthalmic goiter there are the symptoms of that disease. When due to other causes there is generally a tachycardia lasting as long as the palpitation lasts. Other symptoms depend upon the cause. If due to gas distending

the stomach and pressing upward upon the diaphragm, there will be eructations with relief from the palpitation. In anemia there will be an anemic bruit at the base of the heart. Shock and fright will leave the face pale; in excitement the face will be flushed. There may be nervous or hysterical manifestations, dyspnea, etc., attributable to the cause of the palpitation. If there is no organic lesion, the physical signs may be negative, perhaps nothing more than increased frequency or force of heart action.

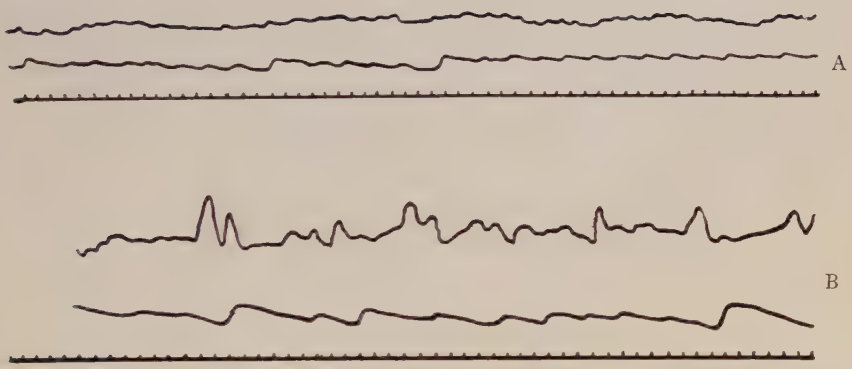
Treatment.—The treatment of palpitation of the heart depends upon its cause. If it is distressing and the cause cannot be removed, an ice bag over the heart and 15 grains of bromide of sodium will often give relief. If there is considerable mental agitation, 5 to 10 grains of veronal and 5 grains of monobromated camphor should be given. If these do not give relief a hypodermic injection of $\frac{1}{8}$ grain of morphine and $\frac{1}{120}$ grain atropia should be used. In all cases the cause must be removed if possible. In many senile cases the cause can be traced to a distended stomach and rapidly acting cathartics are required. In high altitudes the patient should practice rapid and deep breathing. Drugs are rarely required except during a severe attack.

Bradycardia

A pulse rate of 50 to 60 a minute is natural to many aged individuals and is generally due to increased arterial tension, the heart acting slower but more powerfully to overcome the increased resistance of the vessels.

Etiology.—The most frequent cause of bradycardia in the aged has just been stated. It may also occur in organic heart disease, more especially in cardiac degenerations, irritation of the vagus, in various toxemias, in convalescence from exhausting diseases, in chronic diseases, in exhaustion, inanition, sunstroke, syncope, meningitis, apoplexy, etc. As a pure neurosis it occurs with other neuroses and some psychoses, especially with neurasthenia, melancholia, hysteria, epilepsy and paresis. (Heart block, in which bradycardia is a prominent symptom will be described separately.)

Symptoms.—The pathognomonic symptom is a diminished



A. Complete Arrhythmia. Fibrillation of the Auricle (Courtesy of L. F. Bishop, M. D., New York.) *B*. Same case ten days later under influence of Digitalis.

frequency of the action of the heart. The beat should be counted at the heart and never at the radial pulse. Other symptoms belong to the causative condition.

If persistent it is a symptom of organic heart disease, irritation of the vagus, exhaustion, convalescence, etc. Temporary bradycardia may be due to syncope, pain, toxemia and mental depression.

Treatment.—The treatment depends upon the cause. It is rarely necessary to institute treatment for the bradycardia itself, as it gives no distressing symptoms and it will disappear or improve with the arrest or improvement of the cause.

Tachycardia

Rapid heart action is sometimes natural to the individual and cases have been reported in which there was a heart beat of 115 to 130 a minute without distress and without any other symptom or sign pointing to a pathological condition.

Etiology.—In the aged tachycardia is frequently associated with coronary sclerosis. All the causes that may give rise to palpitation may produce rapid pulse. It is present in fevers, goiter, hemorrhage or tumor at the base of the brain, various forms of heart disease and it may be produced by drugs which either stimulate the sympathetic or inhibit the vagus. Tachycardia being a symptom rather than a disease, the pathology depends upon the underlying causative condition. A permanent tachycardia may be physiological, an intermittent one is always pathological.

Symptoms.—Tachycardia is itself a single symptom, rapid heart action. When it becomes noticeable to the patient it is called palpitation. There are usually incidental symptoms belonging to the underlying cause. There is sometimes a precordial distress and in those cases in which it is natural to the individual, slight exertion, or emotion will produce palpitation.

Treatment.—The treatment depends upon the cause. If no cause can be found in permanent tachycardia and the individual is in good health nothing should be done. If intermittent and no cause can be found, the patient should lie down with an ice bag placed over the heart. If the organ is strong

and the sounds are clear, 5-minim doses of tincture of aconite should be given. If the heart sounds are weak, 2-minim doses of aconite combined with 1/50 grain of strychnine should be used. Tincture of digitalis is useless as its action is too slow. Gelsemium or veratrum viride may be substituted for aconite in the same dose. The treatment of palpitation also applies to tachycardia.

Adams-Stokes Disease

This is a form of transmittory arrhythmia in which epileptoid attacks occur at irregular intervals.

Etiology.—It occurs most frequently in cases of arteriosclerosis, occasionally in cases having a history of syphilis or rheumatism. Neither the cause of the disease nor that of the attacks is known; the attacks have occured when the patient had been at complete rest or even in bed for several days and also after slight or intense excitement.

Pathology.—In most cases a lesion in the bundle of His has been found, but pathologists have reported autopsies of cases that had shown the symptoms of the disease, without finding a lesion which could stand in a causal relation to it. On the other hand McElroy reported a case that on autopsy showed almost complete destruction of the bundle of His by a gumma, yet there was no symptom or evidence of the disease during life.

Symptoms.—Before the first attack and between attacks a bradycardia may be the only symptom pointing to the disease and the patient himself may be unconscious of it. There is usually a jugular pulsation or wave much more frequent than the heart beat, the usual relation of the two being three to one. The attack comes on suddenly with tinnitus, vertigo and syncope, the pulse being extremely slow and weak, lasts a few minutes and the patient recovers feeling weak and as though he had just escaped death. It is probable that many cases of sudden death in the aged are due to an attack of this disease.

Treatment.—The treatment is entirely symptomatic. Where a cause can be found, that cause should receive attention. The usual hygienic measures applicable to arteriosclerosis should be employed. During the attack a hypodermic

injection of atropia 1/120 grain and spartein 1/4 grain should be given. Digitalis is contraindicated but strychnine in 1/30-grain dose can be given.

Arrhythmia

Irregularity in rhythm is frequently found in the aged, generally in connection with organic heart disease. The irregularity may be in rate or force or both, temporary, prolonged or permanent. As the disturbance in cardiac action is transmitted to the radial artery, thereby affecting the character of the pulse, the various forms of arrhythmia have been designated by the type of pulse, paradoxical, bigeminal, trigeminal, respiratory, extrasystolic, alternating, etc. As the radial pulse in the aged is, however, affected by many factors besides the action of the heart and is therefore unreliable for diagnostic purposes, the terms usually applied should not be used to designate the character of the arrhythmia.

Complete Arrhythmia.—In complete arrhythmia there is a disturbance in the force and rate of cardiac action, without periodicity or regularity of sequence. In the sphymographic tracings no two successive beats are alike. This condition is found in auricular fibrillation, complete loss of compensation, advanced exophthalmic goiter and may be produced by digitalis or thyroid extract. The irregularity may be extreme producing delirium cordis or it may be so mild as to be unnoticed and unknown until an examination of the heart is made.

Partial Arrhythmia.—In incomplete or partial arrhythmia there is a periodicity or regularity of sequence in the irregularity of rate, or force or both. It may be physiological or pathological.

In *physiological* or *respiratory arrhythmia*, the beats are accelerated and stronger during inspiration and slowed with expiration. Normally the difference is very slight.

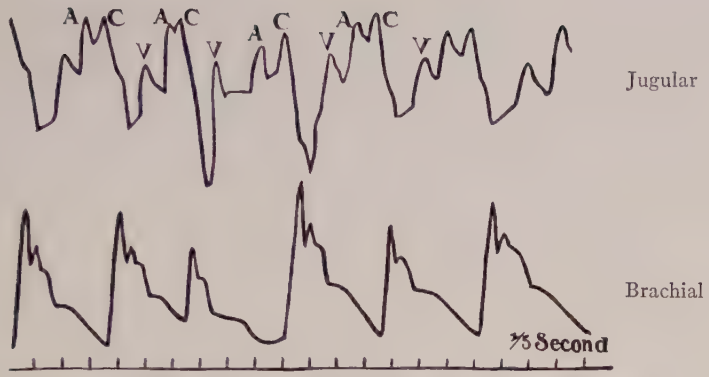
Exaggerated respiratory arrhythmia, the sinus arrhythmia of Mackensie, in which the difference becomes marked, occurs in neurasthenia, convalescence and in cerebral diseases. The force may be lessened when the rate is increased during inspiration, as in pericarditis and weak heart. The pulse is then called paradoxical pulse. Exaggerated respiratory arrhythmia

is supposed to be due to a change in the irritability of the vagus center in the medulla and can be controlled by atropia. The arrhythmia is increased by forced breathing and diminished by holding the breath.

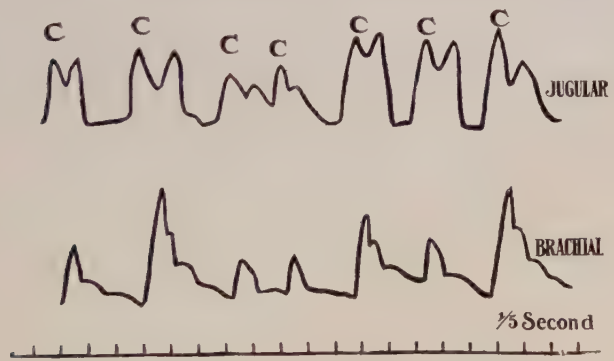
Extrasystolic Arrhythmia.—In this form there is a second systolic beat rapidly following the first or normal systole with a consequent prolonged diastole. Meltzer devised the formula that the diastolic pause between the normal and extrasystole plus the diastolic pause following the extrasystole equals two systoles. The extrasystole is not a supernumerary but an accelerated beat since the following contraction occurs in its normal time. There may be a second or even a third accelerated systole but true to Meltzer's formula the succeeding diastole will be lengthened so that the sum of the diastoles will equal the sum of the normal diastoles. This form of arrhythmia gives rise to the bigeminal, trigeminal, etc., forms of pulse.

The studies of Traube, Gaskell, Mackensie, Wenckebach, Meltzer and others have cleared up many questions as to the origin of the extrasystole but the nature of the stimulus which produces it in man is still unknown. It occurs most frequently in hysterical and neurasthenic individuals, occasionally in arteriosclerosis. The diagnosis of this form of arrhythmia is not difficult, there being two, rarely three or more, beats following each other rapidly and then a correspondingly long pause. The patient may feel a hard thump or heart beat corresponding to the extrasystole (which is usually louder than the normal first sound), others may feel a momentary faintness as though the heart stopped, corresponding to the prolonged diastole. The diagnosis can be confirmed by the sphymograph and cardiograph.

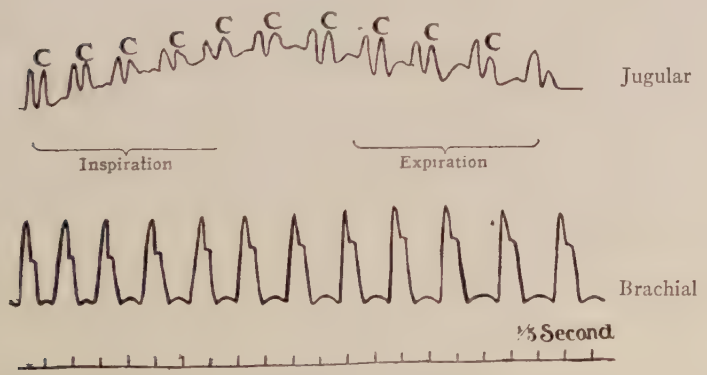
Transmittory Arrhythmia.—This form of arrhythmia to which the name heart block has been given is caused by a discrepancy between the auricular and ventricular contractions due to impaired conductivity of the impulses through the bundle of His. In moderate disturbance, there may be only a retardation of impulses causing an occasional loss of a ventricular beat. If the impairment is greater, frequent beats are lost either irregularly or at regular intervals so that every second or third beat is dropped. Still further disturbance may cause only one out of every two or three impulses of the auricle to reach the



Extrasystole. (Tasker Howard, M. D., *New York Medical Journal*, May 3, 1913.)



Auricular Fibrillation. (Tasker Howard, M. D., *New York Medical Journal*, May 3, 1913.)



Sinus Arrhythmia. (Tasker Howard, M. D., *New York Medical Journal*, May 3, 1913.)

ventricle or there may be complete dissociation between auricle and ventricle, the latter following a rhythm of its own. This is termed complete heart block.

The impairment of the bundle of His may be due to senile degeneration, syphilis, toxemias, or it may be of neurotic origin. The independent rate of contraction of the ventricles is from twenty-three to twenty-eight per minute; if the heart beat does not exceed this number there is a complete block. In Bishop's case in which there was a calcareous nodule in the region of the bundle of His the heart rate ranged from thirty-eight and forty on the first day to twenty on the last day. When transmittory arrhythmia is associated with epileptiform attacks it forms Adams-Stokes disease.

Alternating Arrhythmia.—This is the pulsus alternans first described by Traube, in which the rate is regular but the force is irregular. There may be several strong beats followed by a weak beat, or strong and weak beats alternating or several weak beats with an occasional strong beat. The contractility of the muscle is impaired either permanently through degeneration of the myocardium or dilatation, or temporarily through acute disease or drugs.

Galloping arrhythmia is a form of arrhythmia in which a third sound is heard with each beat of the heart. It is a reduplication of one of the normal sounds usually the second and occurs immediately after the second sound or just before the first one, shortening the diastole. The sounds are suggestive of the hoof beats of a galloping horse. It may occur temporarily when the heart is overworked; if permanent it is generally due to the exaggerated rebound of hypertrophy with aortic arteriosclerosis.

Embryocardia is a form of arrhythmia in which the first sound and the diastole are shortened, the rhythm being like the ticking of a clock. It is called the tick-tack heart and is heard when compensation is lost and in any condition leading to collapse.

Treatment.—The treatment of the arrhythmias depends upon the cause. Only two types, delirium cordis and Adams-Stokes disease require emergency treatment during an attack, since they produce distress apart from the causative condition. In delirium cordis relief can usually be obtained from a hypo-

dermic injection of atropia 1/100 grain and strychnia 1/100 grain combined with either 1/200 grain aconitine 1/100 grain strophanthin or digitaline or 1/100 grain nitroglycerin, the selection depending upon their indications, high blood pressure and full rapid pulse demanding aconite, low pressure and weak rapid pulse requiring strophanthus or digitalis; high pressure and slow weak or irregular pulse requires the nitrites. The nitrites should not be used if the face is flushed, digitalis should not be given if it has been used for a long time, and aconite should not be used if the pulse and heart beats are weak. In some cases the heart instead of pitching about with varying force and frequency will give an occasional jump like the expiring efforts of a fish out of water. This portends a speedy dissolution. Spartein in gr. 1/2 to gr. ii doses may momentarily strengthen the heart increasing the force and frequency of its contractions.

(The treatment of Adams-Stokes disease is given under that head.)

Extrasystole is sometimes due to an overloaded stomach pressing upward upon the diaphragm, thereby disturbing the heart. To empty the stomach by an emetic or the bowels by a rapidly acting cathartic is the first indication in these cases.

If no cause for the arrhythmia can be discovered the treatment must be symptomatic. Rest and the avoidance of coffee, tea and tobacco are imperative. The greatest care must be taken in the selection of drugs.

If there is considerable mental agitation 5–10 grains of veronal combined with 3 grains of camphor monobromate will relieve this condition.

The ordinary hygienic measures rest, diet, hydrotherapy, massage, freedom from excitement, care of the bowels, etc., are necessary adjuncts.

ANGINA PECTORIS

Angina pectoris is a paroxysmal neurosis occurring most frequently in connection with coronary or aortic arteriosclerosis or aneurysm. It also occurs in myocardial degeneration, endocarditis and other cardiac diseases. In some cases no patho-

logical causative factor can be found. Sometimes there is a history of gout, diabetes, syphilis, renal or hepatic disease, but no causal relations between them has been discovered. Occasionally the symptom complex occurs without the cardinal symptom of paroxysmal, precordial pain; this "angina sine dolore" is supposed to be of neurotic origin although any one of the above-mentioned causal conditions may be present. In functional angina pectoris, misnamed pseudo-angina, the underlying cause seems to be a toxin or a neurotic condition, usually hysteria.

Etiology.—Many theories have been advanced to explain the pathogenesis of angina pectoris yet none will apply to all cases. It is evident that there is more than one underlying factor or else there are several conditions giving the same clinical manifestations. One theory is that it is due to sudden increase in tension in the ventricles. Vaso-dilators diminish cardiac tension and they generally give relief, but in some cases they aggravate the attack. The theory that it is due to myocardial ischemia caused by sclerosis or spasm of the coronary artery may apply to some cases, but patients have died during an anginal attack, yet upon autopsy no coronary disease was found. The toxin theory that the disease is due to toxemia holds good in but few cases. Heberden's theory that it is a cramp or spasm of the heart, brought on by an irritation of the heart muscle, does not hold good in these cases in which there is no change in the force or rhythm of the heart. McKenzie's theory that it is due to an impairment in the contractility of the heart is objected to for the same reason.

One pathological condition is almost invariably present in angina pectoris—the stomach is dilated with flatus which is expelled at the moment that the attack ceases. The intimate relation between the stomach and the heart through the pneumogastric nerve will explain why gastric disorders are frequently reflected in cardiac irritation. An overdistended stomach pressing upward upon the diaphragm causes direct irritation of the heart. In the normal, healthy heart this irritation causes arrhythmia, palpitation and precordial pain and often gastric asthma. If the heart is degenerated, this irritation causes either a spasm of the heart or the terminals of the vagus are irritated and they produce the characteristic agonizing pain by contract-

ing the entire organ, or a limited area of the organ, at the same time compressing the sensory nerve endings. This would explain the most frequent cause of angina pectoris. Fear, shock, anger, etc., may act through reflex action upon the vagus, while tobacco and other toxins are direct irritants of the nerve. In all these cases irritation of the vagus is the underlying or basic etiological factor. The true angina pectoris occurs most frequently in those past middle life and these generally have aortic or coronary arteriosclerosis and perhaps myocardial degeneration, and in addition there are probably changes in the intrinsic ganglia of the heart and in the pneumogastric and phrenic nerves due to impaired nutrition. This would account for the greater frequency, severity and danger of angina pectoris in the aged. In younger persons the functional angina due to neurotic or to toxic causes is the more frequent one.

Symptoms.—The cardinal symptoms are an intense paroxysmal pain over the heart, a sense of faintness and an agonizing fear of death. The heart feels as if it were suddenly cramped or crushed and the patient will either be afraid to move or he will clutch at his chest as though he would grasp the heart. The pain is sometimes localized, more often it extends to the neck and goes down the left arm. In some cases the pain seems to involve the whole chest, back, neck and arms. The face becomes pale and ashy colored, there is a cold sweat and in some cases there is the "facies Hippocrates" which is seen just before dissolution. Death sometimes occurs during the attack or in the syncope following the attack. In some cases there is arrhythmia or a feeble fluttering heart, in some there appears to be no change in the force or rhythm while in others there may be palpitation with increased force in the pulse.

In some cases there is a wheezing respiration and dyspnea, a further evidence that the vagus is disturbed. The attack ends with the expulsion of gas from the stomach. The whole cycle lasts a few seconds, rarely minutes. In the functional angina pectoris the attack is not so severe and it is usually prolonged, lasting several minutes. The pain does not radiate to the neck, there is generally arrhythmia, syncope is frequent, but death during an attack is rare. These attacks usually end with the excretion of a large quantity of urine. Hysteria is a

prominent factor in many of these cases. A toxic form of angina pectoris is brought on by excessive smoking or tea or coffee drinking. In this form the pains are spasmodic, lancinating and may come on intermittently for hours. Palpitation, dyspnea, nausea, syncope, occasionally trembling and profuse sweating are the symptoms encountered in this form of the disease.

In the "angina sine dolore" there are the sudden sense of faintness and fear of death, with a precordial ache or distress but without the agonizing pain of true angina pectoris. Like the latter it lasts but a few seconds and is usually followed by eructations of gas. There is usually arrhythmia and dyspnea.

Diagnosis.—True angina pectoris cannot be mistaken. The functional angina gives a causal history, the attacks are milder, longer and more frequent. A mistake may be made if the patient is seen for the first time during a severe attack of functional angina pectoris. If no causal history or history of previous attacks is obtainable it may be necessary to wait until the attack is over before a definite diagnosis can be made.

Treatment.—In the treatment of angina pectoris the most important indication is the immediate relief of the attack. In some cases the inhalation of 5 minims of nitrite of amyl will cause immediate subsidence of the pain. It is probable that in these cases there is a spasm of the coronaries producing myocardial ischemia and consequent weakness of the cardiac walls. This allows an increased influx of blood without a corresponding expulsion and increased tension in the cavities. This would substantiate two of the theories advanced to explain the pathogenesis of the disease. In some cases vaso-dilators increase the severity of the attack and we must resort to chloroform inhalations, giving few drops at a time. As quick action is necessary during the attack, if amyl nitrite is not at hand, a hypodermic injection of 1 minim of a 1 per cent. solution of nitroglycerin should be given. If the patient has once had an attack he should be instructed to carry nitrite of amyl pearls with him and as there is usually a prodromal sense of uneasiness in the cardiac region before the attack, he should crush a pearl in a handkerchief and inhale it. The pearls can be obtained in silk bags which can be crushed between the fingers. At such times every second is precious. If

neither amyl nitrite nor chloroform gives relief, we must give a hypodermic injection of 1/4 grain of morphine combined with 1/100 grain of atropia. If there is palpitation an ice bag over the heart is generally of service.

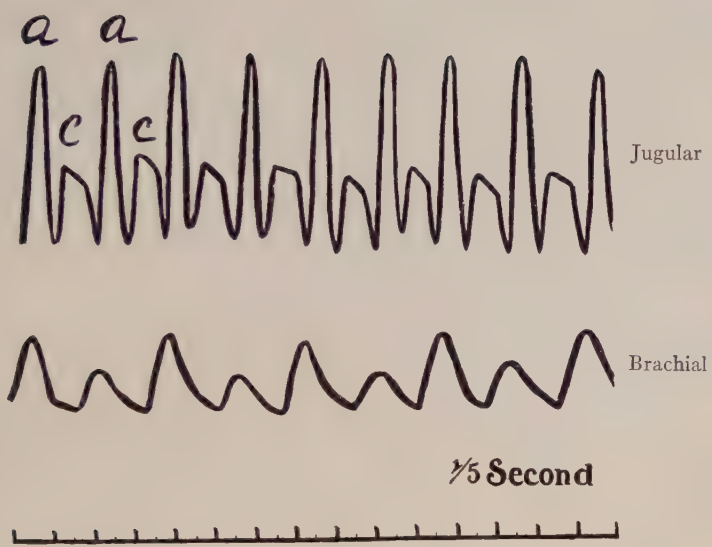
As the attacks occurring in the aged are almost always associated with coronary or aortic sclerosis and cardiac degeneration, the treatment between attacks should be directed to these conditions. In the functional anginas whether neurotic or toxic, the underlying cause must receive attention. Smoking, tea and coffee are injurious in all cases. Excitement and laborious tasks, especially such as require a sudden exertion, must be avoided. Sudden ane powerful emotions have been known to bring on attacks or aggravate the disease by increasing their frequency and severity. In some cases of functional angina we can find no causative factor and we must eliminate everything that might be considered injurious, even tea and coffee though these had been used sparingly; we must avoid all physical strains, including straining at stool, and all sources of excitement.

(For the treatment of coronary sclerosis, see Arteriosclerosis, and for cardiac degeneration see chapter on Cardiac Degenerations.)

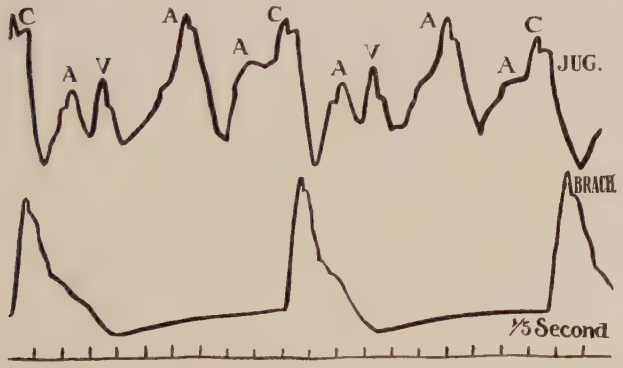
SENILE BRONCHITIS

This form of bronchitis is an atrophic catarrh of the degenerated mucous membrane of the air passages. It is a purely senile condition.

Etiology.—It occurs most frequently in those who live in houses heated by hot air or where no provision is made to keep the air humid. They rapidly develop an atrophic state of the mucous membranes with diminished sensibility and lessened secretion. Dust collects upon this mucous membrane and owing to the diminished sensitiveness, the dust does not create the sensory irritation necessary to produce cough which would dislodge it. The expired air is not expelled with sufficient force to carry off the deleterious substances with which the lining membrane of the bronchi is coated and they produce a constant irritation of the membrane with increase in the flow of mucus.



Alternating Arrhythmia. (Tasker Howard, M. D., *New York Medical Journal*, May 3, 1913.)



Transmitting Arrhythmia. Partial Heart block. (Tasker Howard, M. D., *New York Medical Journal*, May 3, 1913.)

Pathology.—There are the usual senile changes in the bronchial tubes, atony and waste of the muscular fibers, atrophy of the mucous membrane which becomes loose and flabby with diminished sensitiveness and waste of the ciliated epithelium. The glands are atrophied but there is a slight flow of thin mucus mixed with epithelium, leucocytes and dust. The mucus in the finer bronchioles may be thick and tenaceous. The tubes are coated with dust imbedded in mucus.

Symptoms.—The principal symptom is a morning cough by which a small amount of mucus is brought up, usually after considerable effort. The mucus is thick, dark, tenaceous and free from pus. It contains epithelium, leucocytes, pigment granules, dust, etc. The patient does not cough during the day unless he has made some great effort in which the lungs were used excessively, as in shouting or much talking, or if there has been excessive irritation as by inhaling irritating vapors, or entering a very dusty apartment. Certain forms of dust or vapor may be more irritating than others, as tobacco smoke, the vapor of roasting coffee, pollen, etc. In such case the cough may be paroxysmal, very severe and exhausting yet yielding nothing more than a drop of inspissated mucus.

The physical signs of senile bronchitis are in evidence before the morning cough but not afterward. The first few respirations—after the patient has arisen and the level of the mucus has been changed—will bring out moist râles, heard best in the lower part of the chest near the spinal column where the mucus had collected by gravitation. After the mucus had been coughed up the chest is free from râles. Percussion may reveal a duller note where the mucus had accumulated.

This form of bronchitis is differentiated from other forms by the scanty secretion, absence of signs after the morning cough, absence of temperature, absence of pain except when a paroxysmal cough is induced during the day, its persistence and its occurrence at any time of the year. In senile emphysema râles are heard in the back immediately upon arising but they are sibilant or snappy caused by the opening of the air vesicles which had been compressed while the patient was in the recumbent position. There is no cough in this condition.

Treatment.—Senile bronchitis may be relieved by medicinal measures but a cure can be effected only if the cause is removed.

A dense humid atmosphere free from dust and vapors, and an equable climate are imperative. This can best be obtained near the seashore either in Florida or southern California. High elevations should be avoided. The activity of the mucous glands should be stimulated and for this purpose nothing equals the syrup of the hypophosphite of ammonium, given in dram doses every four hours.

Menthol and eucalyptol inhalations are stimulating and may be used for the upper air passages. If the secretion is thick and tenaceous the muriate of ammonia should be given in 5-grain doses three or four times a day and if there is any difficulty in expectoration, senega, ipecac, squills or similar expectorants may be tried.

Morphine, codeine, atropine and other drugs which diminish the secretions are contraindicated. Spasmodic attacks of coughing can generally be relieved by the bromides, preferably the bromide of ammonium.

SENILE GASTRIC CATARRH

The terms senile gastric catarrh, chronic gastric catarrh and chronic gastritis, when applied to the senile degeneration of the stomach, are misnomers as there is neither a catarrhal nor an inflammatory process. Ewald declares that there are no exclusively senile gastric or intestinal diseases. This is true to the extent that the symptoms of senile catarrh may appear in earlier life and that similar anatomical and physiological changes as occur in senility may occur as pathological conditions earlier. When we consider, however, that these pathological conditions of maturity are physiological conditions in old age and that the altered functions in old age are the normal functions at that period of life we must consider the hyperactivity, hypoactivity or perversion of these functions as true senile disorders. As long as the manifestations of senility are looked upon as symptoms of a pathological condition of maturity, so long will there be opposing views as to the nature, character and treatment of diseases that appear as changes from the normal senile state. The condition here described as senile gastric catarrh is one of these diseases. (The chronic gastritis which follows the acute inflammation of the stomach will be described with acute gastritis.)

Etiology.—Perversion of the normal function of the senile stomach may occur without any apparent cause. In most cases it is due to overfeeding or to too frequent feeding through failure to recognize the diminished need of the organism for food and the slower gastric digestion. In some cases there occurs fermentation or decomposition in the stomach, especially if meats or eggs are taken that had been long in cold storage, or if beer is taken with the meals. Gastric fermentation is less injurious than gastric decomposition since toxins are elaborated in the latter process and are absorbed. Excessive amounts of protein may remain for many hours in the stomach and if additional food is introduced before the residue has been disposed of, food will constantly be present in the stomach in various stages of digestion, exhausting the organ. Irritating substances will produce an acute catarrhal or inflammatory condition, not the chronic condition here described. Improperly masticated food may produce either the acute or the chronic condition.

Pathology.—There are the ordinary senile changes, atony and waste of muscular fibers permitting a dilatation of the organ; thinning of the mucous membrane and atrophy of the glands; diminution in peptic secretion and in the amount of hydrochloric acid. The pyloric sphincter is sometimes hypertrophied; occasionally there is atony permitting dribbling into the duodenum of undigested or partly digested food that should have been prepared and converted in the stomach. After prolonged irritation from the etiological factors mentioned, the mucous membrane undergoes granular degeneration and may disappear almost entirely. In extreme cases of senile atony the stomach is little more than a reservoir for food, with slight peristaltic power and little digestive capacity. In the latter case, the digestive work is done by the intestines and as long as they are able to carry on this work the nutrition of the organism will continue unimpaired. When the intestines fail the grave results of inanition quickly follow.

Symptoms.—The symptoms of senile gastric catarrh are for the most part exaggerations of the normal senile manifestations and coming on slowly and gradually they are not noticed by the individual until a pathological condition has been produced or secondary symptoms appear. The earliest of the primary symptoms is anorexia. The appetite is normally

diminished in the aged and if the senile changes are far advanced the appetite may fail altogether. There is a sense of fulness in the stomach lasting sometimes for hours after a meal. Flatulence and eructation of gas are frequent accompaniments of this condition and if the stomach is dilated with gas, it may press upward upon the diaphragm, disturbing the heart action and producing the syndrome called *gastric asthma*. If this occurs there is palpitation or arrhythmia of the heart, dyspnea, and if severe there may be vertigo, syncope and even collapse. The cases of sudden or rapid death from acute indigestion are cases where the rapid and excessive formation of gas in a dilated stomach caused sudden disturbance of the heart action with consequent paralysis of the heart or interference with the cerebral circulation. Vomiting is rare except when the stomach is overloaded with food and even then some extraordinary irritation is required to arouse a sufficiently powerful reflex action to cause vomiting.

Cabot says "any type of dyspepsia, any sort of genuine gastric trouble occurring in a person over forty years, who has never had any such trouble before, is strongly suggestive of cancer." After the age of sixty senile gastric catarrh occurs far more frequently than malignant disease, and Cabot's statement should apply only to the period between forty and sixty. It is sometimes impossible to make a positive early diagnosis of carcinoma of the stomach, as the earliest symptoms resemble the early symptoms of senile gastric catarrh, there being in both gastric dilatation and hypoacidity. As vomiting is rare in the senile form of dilatation which is due to atony and it is an early symptom of dilatation due to obstruction of the pylorus, this may serve to differentiate cancer from senile catarrh. If vomiting does occur in senile catarrh it is due to excessive or improper food and the substance brought up is food in various stages of digestion but there is little mucus and no blood. The vomited matter in cancer almost always contains mucus and often blood. The later symptoms of carcinoma are sufficiently distinctive to prevent an error in diagnosis. The absence of pain in senile gastric catarrh is a strong diagnostic point for the elimination of ulcer, cancer and acute and chronic gastritis. Senile gastric catarrh can be differentiated from chronic gastric catarrh which is secondary to the acute form by the absence or

small quantity of mucus which is brought up, while in the chronic or secondary form a large quantity of mucus is vomited. There is also a history in the case of the secondary form which determines the underlying cause.

Treatment.—The treatment of senile gastric catarrh comprises dietary and hygienic, medical and mechanical measures. The first indication is to clean out the stomach. Some authorities say lavage is easily accomplished in the aged owing to lessened sensibility of the pharynx, esophagus and stomach. On the other hand spasm of the muscles of deglutition and of the glottis is easily induced and a fatal asphyxia may result. To prevent spasm a spray containing 2 per cent. of cocaine should be used in the throat and a stomach tube of small caliber should be employed. A 3 per cent. solution of boracic acid can be used to wash out the stomach. After lavage the stomach should be given a rest for two hours, after which give 1/50 grain of strychnine or 5 minims of the tincture of nux vomica combined with a dram of compound tincture of gentian or colombo and the same amount of water. Food can be given ten or fifteen minutes later. Excellent results have followed the foregoing plan of treatment. Lavage can be employed every day for three or four days, then every second or third day. The food should be concentrated containing little meat and little cellulose. Predigested foods are recommended but most of the foods of this character contain a large percentage of alcohol. The patient may take meat juice, soft boiled eggs, cream, malted or evaporated milk, toasted bread, well-boiled vegetables containing little cellulose, etc. Water acidulated with hydrochloric acid should be taken during the meal. Ewald recommends that the water should be as strongly acid as the patient can swallow without difficulty and it should be taken after meals. As liquid introduced into the stomach filled with food does not mix with the food but passes off into the duodenum, the advice to take the acidulated water after meals is irrational. Pepsin should be given with or immediately following the meal. If there is much flatulence charcoal in 5-grain doses should be added to the pepsin. Incidental measures are the simple bitters like gentian, colombo, cinchona and quassia in dram doses of the tincture or 5 grains of orexine for the anorexia, and nux vomica as a tonic and a glass of hot water

containing a teaspoonful of common salt or phosphate of soda upon arising. This washes away the mucus with which the inner surface of the stomach becomes coated during the night, and it also acts as a mild laxative.

It is sometimes possible to relieve a gastric catarrh by these means alone if the stomach is then given complete rest for the day and the saline hot water is repeated at bedtime. The diet must thereafter be selected to give the stomach as little work as possible and to produce as little gas as possible. We must warn again against meat, fish and eggs that have been kept long in cold storage or have been preserved with chemicals. Nausea and vomiting are rare and if vomiting occurs it is nature's method of getting rid of offending material. If there is nausea and the stomach is filled with food, 5 grains of pepsin should be given to aid digestion and 1/4-grain aloin to stimulate stomach peristalsis. If the nausea occurs when the stomach is empty it may be due to an accumulation of mucus in the stomach. Occasionally nausea upon arising will be caused by the accumulation of mucus in the pharynx. In either case hot water containing a small quantity of salt should be taken to dislodge the mucus. A persistent nausea when the stomach is empty is rapidly relieved by 1/12 grain of cocaine hydrochlorate. This will also relieve gastrodynia. Pyrosis is infrequent in the aged but if it does occur 5 grains of bismuth subnitrate combined with either 1/12 grain of cocaine or morphine should be used. If hyperacidity occurs it is due to acetic, butyric and lactic acids, all decomposition products. Alkalies which cure hyperacidity in maturity are contra-indicated in the aged and we must resort to antifermentives like salicylic acid, boracic acid, creosote, charcoal, etc., to prevent further fermentation and decomposition.

The most important hygienic measures are freedom from worry, mild exercise and regulation of the bowels. A temporary constipation may undo in two days, all the good obtained by several weeks' treatment. Salines should be given either in an occasional large dose or in small doses for several days. They should not be given in habitual constipation. The ordinary senile constipation should be treated as indicated in the chapter on Senile Degeneration of the Intestines.

GASTRIC NEUROSES

Gastric neuroses occur rather frequently in the aged.

While some of these diseases are rare in the aged and others are apparently associated with hysteria or neurasthenia, they are all placed in the second group of diseases upon the assumption that most of the neuroses occurring in the aged are due to the senile change in the nervous system, to arteriosclerosis or change in the stomach or its secretions.

Pneumatosis or distention of the stomach with gas which the stomach cannot dispel owing to atonicity of the walls is of frequent occurrence. It is one of the causes of gastric asthma, the dilated and distended stomach pressing upward upon the diaphragm and thus upon the heart. The treatment of this condition is the treatment of gastric atonicity. If rapid eructation of gas is necessary 10 to 20 minims of oil of turpentine should be given and pressure applied over the stomach. Turpentine stupes over the abdomen will give temporary relief. In some cases 5 grains of willow charcoal and 5 grains of sodium bicarbonate will be more effectual than the turpentine. To prevent excessive fermentation dilute hydrochloric acid should be given with every meal. Nervous eructations are infrequent in the aged. They may occur when food is taken in too rapid intervals.

Pyrosis or heartburn is usually due to gastric dilatation with hyperacidity. Hyperacidity being, however, rare in the aged, pyrosis is also rare. When it does occur, the underlying condition must be treated. For immediate relief we can give 1/8 grain of cocaine.

Grastrospasm either at the cardiac or pyloric orifice, is occasionally met with in old age. It sometimes follows a cold drink, a strong alcoholic beverage, sharply spiced food or strong emotion. Pyloric spasm may follow an excessive meal or food improperly or insufficiently masticated. In some cases no cause can be discovered. The treatment depends upon the cause, if discoverable. In other cases abstinence from food will frequently give relief. The bromides are useful in this condition.

Relaxation of the pylorus occurs occasionally from atony of the pyloric sphincter. (This is described under the Senile Degeneration of the Stomach.) Other causes are shock or

strong sudden emotion such as fright, etc. A persistent relaxation may be suspected when there is a lientery containing meat fibers. The treatment depends upon the cause. Nothing can be done for the temporary condition due to shock, fright or similar causes. *Supermotility* does not occur in the aged.

Secretory neuroses are infrequent in the aged. *Hyperchlorhydria* and *gastrosuccorrhea* are extremely rare and *hypochlorhydria* as part of an *achylia gastrica* is a normal condition in the aged, due to atrophy of the secreting glands. This is not a neurosis but a true senile degeneration for which nothing can be done. Where it exists we can supply the deficiency artificially.

Sensatory neuroses occur rather frequently, the most prominent being *anorexia.*

Diminished appetite is natural in the aged. Diminished activity causes less waste and less expenditure of energy, and less food is consequently required. Other factors which tend to diminish the appetite in the aged are lessened salivary secretion, dysphagia, and some change in the taste bulbs. Nothing need be done for this but if there is complete anorexia, the appetite must be stimulated by means of simple bitters. Orexine is especially useful in this condition. It is given in 10-grain doses about an hour before meals.

Bulimia does not occur in the aged except as a symptom of diabetes, and occasionally during convalescence from prolonged illness. It requires no treatment. The elixir of the valerianate of ammonia has been found to diminish the appetite but it is rarely necessary to give it.

Parorexia or perverted appetite occurs occasionally where the taste for ordinary foods is lost. This is generally due to gustatory perversion, and manifests itself in a malacia, the patient craving spiced, acid or acrid foods. The craving for indigestible substances such as occurs in hysteria does not occur in the aged unless associated with hysteria, dementia or other psychosis. The painful gastric neuroses are rare in the aged.

Hyperesthesia may occur during neurasthenia or after a shock, fright or other strong emotion but it does not last longer than a few hours. Suggestion or autosuggestion may, however, cause its reappearance and from this cause it may become per-

manent. In this as in other painful neuroses psychic measures will often avail while drugs will be useless.

Gastralgia or gastrodynia is another painful neurosis which may be due to suggestion. It is generally associated with neurasthenia, the attacks being most severe when the individual is most depressed. The pain comes on independently of food and in some cases taking food gives relief. Gastralgia resembles a colic, moderate pressure giving relief while deep pressure will uncover a point of tenderness. Hepatic colic is more painful and the tender point is usually to the right of the sternum and umbilicus; intestinal colic is more diffuse and not over the stomach; renal colic has also a pathognomonic site between the kidney and the bladder. Cancer and ulcer are readily differentiated from simple gastralgia. Gastric ulcer is rare in old age and it can be distinguished by the presence of a hyperchlorhydria. In cancer the history, cachexia, vomiting, localized pain and later the presence of a growth ought to differentiate it from gastrodynia. Intercostal neuralgia is more severe, is higher up and more localized.

In the treatment of the painful senile neuroses, psychic measures will often avail. When drugs are required bromide of sodium or strontium should be used and in an emergency for the more rapid relief of pain we can use cocaine or eucain. The narcotics should not be used. Acidulated waters are generally well borne. Regulation of the diet will in some cases completely cure a neurosis, and in some cases a day's starvation will cause the symptoms to disappear.

Esophageal Neuroses.—*Spasm* of the esophagus occurs occasionally in the aged either as part of a general neurosis, such as hysteria or as the result of local irritation as when a hard substance is swallowed. In some cases there is a prolonged contraction of the esophagus, food failing to pass a certain point for several minutes or hours, then passing without difficulty. In some cases certain articles of food, solids or liquids cause spasm. As the underlying cause can seldom be discovered, the treatment must be symptomatic, substances causing spasm must be avoided and bromides should be given. Galvanism, faradization, or fine rapid vibration sometimes gives relief. Psychic measures are often effective. The patient may have a spasm through fear and if the fear is allayed the

spasm will not occur. In one case in which swallowing water produced a spasm, a glass of water slightly flavored and given by the physician as medicine was swallowed without difficulty.

Globus hystericus, anesthesia and hypesthesia are usually hysterical phenomena and need no other treatment than that for the underlying condition.

Intestinal Neuroses.—*Intestinal neuralgia* is extremely rare and is supposed to be due to arteriosclerosis of the abdominal aorta or the mesenteric artery. *Colic* is almost invariably due to the presence of an irritant or peristaltic stimulant. Excessive motility does not occur except when due to a stimulant and atony is almost always due to the natural senile degenerative changes in the walls. The treatment depends upon the cause.

CHOLELITHIASIS

Gall-stone formation is the most frequent disease of the liver and its adnexa in old age, and autopsies frequently reveal gall-stones in the gall-bladder, though often they gave no indication of their presence during life.

Etiology.—The frequent finding of gall-stones in the aged at autopsy, which gave no symptoms during life, would tend to exclude infection as the principal etiological factor and would point to a change in the character of the hepatic secretion or to diminished expulsive activity of the gall-bladder, probably both, causing an increase in the proportion of cholestein, sometimes a deposit of calcium salts, and stasis. The most prolific causes in earlier life, infection and inflammation of the gall-bladder may prevail in old age, but infection is rare, while inflammation is more often resultant than causative. In MacCarty's statistics nearly 70 per cent. of cases of acute cholecystitis had gall-stones and 93 per cent. of chronic cholecystitis cases were associated with gall-stones. As acute gastrointestinal catarrh is infrequent in senile cases and chronic atrophic catarrh has not the tendency to extend through the common duct to the gall-bladder this cause of cholecystitis is rare, and we can assume that if cholecystitis is present it is due in most cases to the irritation produced by the concretions.

Symptoms.—As has been stated above, many cases give no symptoms. In some cases the only suspicious symptoms are

those, connected with deficient bile supply; clayey, foul-smelling stools, perhaps containing fat globules, a yellowish furred tongue, bad breath and sallowness. If in these cases pressure is made over the region of the gall-bladder and tenderness is elicited there is a mild cholecystitis present probably due to gall-stones. There is no pathognomonic symptom of gall-stones except their presence in the stools. The hepatic colic is caused by the spasmodic contraction of the unstriped muscle fibers of the gall-bladder, in the effort to expel and propel the contents, which may be pus, mucus or blood as well as bile concretions. In senile cases, however, if these morbid contents are present, they are due in almost every case, to infection, occurring especially during typhoid fever. The symptoms resolve themselves into the symptoms of cholecystitis, obstruction of the ducts and colic. In mild *cholecystitis* there is generally a dull ache, not severe enough to produce actual suffering but quite pronounced at times, and often we may elicit pain upon pressure over the region of the gall-bladder, and in the back about an inch to the right of the eleventh dorsal vertebra. In pronounced inflammation the pain is severe and there is fever, either intermittent if due to bacterial infection, or a steady increased temperature if due to local non-bacterial irritation. In rare cases in the aged a cholecystitis exists without cholelithiasis and in these cases the contraction colic is shorter and milder than when gall-stones are present. Jaundice is sometimes observed, and bile is occasionally found in the urine.

When gall-stones lodge in the common duct they give rise to symptoms of *biliary obstruction*. (See *Biliary Obstruction*, page 329.) There is then deficiency of bile supply to the bowels, jaundice and colic. The jaundice is variable, being slight in some cases and severe in others, always deepening after a colic paroxysm. With this form of jaundice we find bile in the urine and none in the feces.

The *colic* which is the most constant and characteristic attendant of gall-stones occurs in paroxysms which are fairly regular when the duct is involved, but occurring at irregular intervals when the gall-bladder alone is affected. In senile cases the attacks are not as severe as in earlier life nor are the chills, sweating and fever accompanying the pain as pronounced. The pain occurs as a sudden agonizing neuralgia in the right

hypochondrium, radiating toward the right shoulder. If the colic originates in the common duct the pain begins in the epigastric region and radiates backward and not upward. In this case the remittent jaundice distinguishes it from other abdominal colics. The diagnosis of cholelithiasis is rather difficult in senile cases, owing to the mildness and irregularity of the symptoms. Old cases may give the characteristic symptoms of hepatic colic but usually the rigor, chill and fever are not severe, there is no vomiting and but little sweating, while jaundice is rare. In some cases there are occasional sharp pangs in the epigastrium and pain can be elicited on pressure over the gall-bladder. More often there is a persistent dull ache in the epigastrium with the symptoms of deficient biliary supply to the bowels. In these cases there may be paroxysms of pain marking the passage of a calculus through the duct and then there is intense mental and physical depression similar to shock. The diagnosis has often been confirmed by radiography.

Complications.—The number or size of the concretions may fill the gall-bladder producing inflammation and ulceration. As a remote result rupture of the organ may ensue, followed by rapidly fatal shock or peritonitis. Other complications are biliary fistulæ, intestinal obstruction by gall-stones, adhesions of the organ, cancer.

Treatment.—During the attack relief from pain is the only indication and for that purpose there is nothing that will take the place of a hypodermic injection of morphine combined with atropine, giving 1/4 grain of morphine as an analgesic and 1/100 grain of atropine to counteract the effect of the morphine upon the respiratory centers and to cause relaxation of the muscular fibers. Chloroform inhalation will also give immediate relief but is dangerous if there is arteriosclerosis. Olive oil in from 2- to 6-ounce doses has been recommended but it is doubtful of having any other effect except that of aiding in the expulsion of those calculi that reach the duodenum. Chloral hydrate is excellent in younger individuals but it is dangerous in the aged.

The treatment in the intervals between attacks depends upon the severity and frequency, the amount of distress and the general condition of the patient between the attacks. If the symptoms are mild and the attacks are infrequent operation is

unnecessary. If there is a fatty heart, myocarditis or cardiac dilatation, nothing but the certainty of death without operation will justify surgical interference. If the attacks are frequent and severe or if there is an infective cholecystitis, operation becomes necessary, sometimes even imperative. In all other cases, however, medicinal measures should be tried before resorting to cholecystectomy or cholecystostomy. There is no known method of resolving gall-stones *in situ*. Numerous drugs have been recommended for this purpose, those most frequently employed being the sodium choleate, sodium oleate, sodium salicylate, sodium succinate and iron succinate, olive oil and oil of turpentine. The salts have the property of stimulating the flow of bile and making it more fluid but it is doubtful whether they have any effect upon the concretions already formed. By increasing the fluidity of the secretion, further formation of calculi is prevented and this often suffices to prevent a recurrence of an attack. The sodium choleate in 5-grain doses twice or three times a day has this effect upon the bile and also supplies the deficiency of the duodenal secretion. The sodium succinate acts more powerfully upon the liver and upon the secretion but has no effect upon foods in the intestinal tract. Drug treatment must be continued for months or years, as their effect is only temporary. If the attacks continue during the drug treatment with undiminished severity or reappear after discontinuance of prolonged drug treatment, operative measures become necessary. The form of operation, whether cholecystectomy or cholecystostomy, will depend upon the exploratory findings and the surgeon's preference.

SENILE METRITIS

Metritis originating after the menopause and not associated with a growth nor produced by traumatism is rare.

Etiology.—Most of the reported cases were due to the retention of mucus through vaginal atresia or cervical occlusion, with subsequent septic infection. A hemorrhagic form, which is, however, extremely rare, is supposed to be due to a cardiac lesion with consequent venous stasis. Various predisposing causes have been suggested but the cause given for the purulent form is sufficiently potent to explain every case of this form.

It occasionally occurs soon after the menopause, more frequently a few years later.

Symptoms.—The earliest and most pronounced symptom is a purulent or sanguinous discharge having a most offensive odor. In some cases the discharge is scanty, in others copious; sometimes intermittent, at other times continuous. There is usually little pain; occasionally a colicky pain in the uterus precedes a sudden gush of the discharge. In some cases there is a rapidly progressive cachexia with emaciation, sallowness, and gastric disorders. In making a digital examination partial atresia of the vagina and a vaginitis are usually found. The cervix is soft, apparently swollen and painful to the touch. Examination with the speculum is frequently impossible owing to the constriction and the inflamed condition of the vagina. The fetid odor and the cachexia cause this disease to be generally mistaken for uterine cancer and cases have been operated upon which in their clinical manifestations could not be distinguished from uterine cancer. A curettage scraping should be examined in every case of doubt. The determination of the actual pathological condition present is of the utmost importance, the life of the patient depending upon the treatment, which is entirely different in the two diseases. If the curette scraping does not clear up the diagnosis or if curettage is impracticable it is better to await the result of treatment for metritis than to conclude that we are dealing with a uterine cancer and proceed to perform a hysterectomy. The disease is grave and while most cases recover under appropriate treatment, there is always danger of spreading local and general infection, and of exhaustion.

Treatment.—The primary indication is to clean out the uterus. The cervix must be dilated and the contents of the uterine cavity should be washed out with a mild solution of permanganate of potash or sterile water. This may be followed by a solution of peroxide of hydrogen. Curettage with a sharp curette is dangerous on account of the thin and degenerated walls, and with a blunt instrument it is useless. Only the necessity of arriving at a correct diagnosis justifies the use of the sharp curette in making a scraping for examination. After the cavity has been emptied and cleaned it should be packed with iodoform gauze. This treatment should be

continued for several days after which it will suffice to pack the vagina alone until the discharge ceases. The constitutional treatment consists of absolute rest in bed, tonics and the usual treatment for exhaustion. The same treatment is indicated in the hemorrhagic form but curettage is positively contraindicated.

CEREBRAL ANEMIA

Cerebral anemia is frequently found in the aged but its advent is so slow that the patient accommodates himself to the symptoms and does not notice them or ascribes them to the result of ageing.

Etiology.—It is generally due to sclerosis of the cerebral arteries with weak heart and especially with aortic stenosis.

Symptoms.—When due to cerebral arteriosclerosis there will be the symptoms of this disease, vertigo, dizziness, tinnitus, weakened memory and neuralgic or prolonged headaches with drowsiness and a feeling of emptiness in the head. The patient will have an instinctive desire to lie down and the symptoms will subside in the recumbent position. In cerebral arteriosclerosis without anemia change of position does not relieve these symptoms. Cerebral anemia due to other causes is readily differentiated from this form which is peculiar to the aged.

Treatment.—The treatment is the same as for cerebral arteriosclerosis. The hypodermic use of sterile solutions of arsenic and iron or the administration of hemoglobin in 15-grain doses three times a day may improve the character of the blood. The symptoms usually increase, however, and syncope may occur after any excitement or even while but taking a hot foot bath. The nitrites and cardiac stimulants are then indicated.

ALTERNATING CEREBRAL ANEMIA AND HYPEREMIA

This is a disturbance of the cerebral circulation in which there is a progressively increasing anemic condition when the patient is sitting or standing and a progressively increasing hyperemic condition when the patient is lying down. This occurs normally in a mild degree, prolonged standing causing a

mild cerebral anemia with consequent drowsiness and sleep. Some cases of anemia develop, however, a pronounced hyperemia in the recumbent position, which is nothing more than an exaggeration of what occurs normally. It is probably due to some defect in the vasomotor regulation and to atheromatous but not calcareous cerebral vessels.

Symptoms.—The patient awakes with a dull frontal headache and mental confusion, flushed face, injected conjunctivæ and the concomitants of cerebral hyperemia. These gradually pass away after arising, sometimes within a few minutes, sometimes in an hour or two. There is then no symptom of cerebral disturbance for several hours when the symptoms of cerebral anemia appear. The face becomes pale, lips and ears are slightly blanched, the patient feels tired and drowsy and there may be vertigo, tinnitus, headache, and unless he lies down, there will be syncope.

Upon lying down these symptoms gradually disappear and are followed by the symptoms of cerebral hyperemia. There is a gradually increasing frontal headache, a feeling of heaviness, mental dulness and an instinctive feeling that he will be relieved upon arising. If he falls asleep, he will become restless after a few hours, snore, moan and will awake with the symptoms of cerebral hyperemia, thus completing the cycle.

Treatment.—As there are two opposing phases of this disease, whatever will benefit the one will be detrimental to the other. The hyperemic is the more serious phase on account of the distress and possible secondary effects. The anemia can be relieved temporarily by the use of the nitrites, giving 1 minim of a 1 per cent. solution of nitroglycerin when the symptoms appear. The patient will instinctively want to lie down and this affords speedy relief. For the hyperemia we need rapidly acting vasoconstrictors like ergot, or digitalin or strophanthin hypodermically. The drug must be stopped as soon as the effect is produced. The patient must lie with the head elevated and upon arising he should take a hot foot bath. Drugs used for the relief of symptoms must be rapidly acting, the effect passing away in a few hours. There is no way of restoring the impaired vasomotor centers. Strychnine arsenate has been of service in some cases. It is given in doses of 1/100 grain three times a day.

CEREBRAL SOFTENING

Cerebral softening is a degeneration of brain substance due to sudden or rapid deprivation of nutrition. It differs from the normal senile degeneration which involves the whole brain, proceeds slowly and has but a diminished blood supply, while in cerebral softening the blood supply is completely withdrawn from a part. It occurs in two forms—the usual senile thrombotic form which comes on gradually and the embolic form which comes on suddenly. While there are other causes than thrombosis and embolism every case can be placed under one of these two heads of gradual or sudden form of cerebral softening.

Etiology.—The most frequent cause of cerebral softening in the aged is a thrombus in an atheromatous vessel of the circle of Willis or in one of the branch vessels. It may also occur in cerebral arteriosclerosis in which the lumen of a vessel is obliterated. An embolus, which is the most frequent cause of cerebral softening in earlier life does not occur as frequently in the aged. Such accidental causes as syphilis, infectious diseases, anemia, leukemia, etc., which may cause endocarditis with vegetations and consequent embolus, or carbonic-acid poisoning, burns, tumors and other local conditions which may cause thrombosis, are rare in the aged.

Pathology.—The first change is an anemia of the tissue supplied by the vessel which is blocked. In from three to four days this tissue begins to soften into a creamy or pale semifluid mass. This exhibits under the microscope débris of neuroglia, altered cells and fibers, a mass of leucocytes attacking the degenerated tissue and disposing of it by phagocytosis. The destroyed area becomes later filled with fibrous tissue.

Symptoms.—The two forms of cerebral softening differ markedly in their onset but after the brain substance has begun to degenerate they are alike.

Thrombotic cerebral softening comes on gradually. For weeks perhaps there were symptoms of cerebral atheroma, with headache, vertigo, nausea and occasional confusion of ideas. As the nutrition of the part becomes diminished there are symptoms of impaired functions. Numbness in one hand and diminishing strength with gradual loss of sensation and

power set in. The face becomes paralyzed on the same side and mental confusion becomes more marked. With complete closure of the vessel the patient becomes unconscious and the whole side is paralyzed. When the patient recovers from the unconsciousness, there is mental confusion, motor paralysis and in some cases aphasia. In a mild case the patient may not lapse into unconsciousness but the other symptoms will appear.

Embolic cerebral softening comes on suddenly like apoplexy from which it is sometimes difficult to distinguish. In some cases there are no premonitory symptoms. The patient suddenly becomes unconscious and upon awaking we find hemiplegia, mental confusion and sometimes aphasia. In another class of cases the attack begins in an agony of fear, followed rapidly by mental confusion, aphasia, clonic spasms of one or both extremities, rapid loss of motion and sensation on one side, followed by unconsciousness lasting fifteen to twenty minutes. Upon awaking there is hemiplegia and aphasia with some mental confusion. Such attacks may occur several times during the following two or three days. Mild cases in which a small branch alone is involved may present the symptoms of thrombosis but the symptoms come on more rapidly.

Blocking of particular vessels gives pathognomonic symptoms. If a vessel on the left side is blocked, aphasia is produced. The anterior cerebral vessels are rarely attacked and they give ill-defined symptoms, as collateral circulation is speedily established. There will be mental confusion and monoplegia of the opposite side, which soon disappears when circulation is restored through the collateral branches. Blocking of the middle cerebral artery produces hemiplegia and hemianesthesia. The branches produce various monoplegias and those on the left side produce in addition various forms of aphasia. The blocking of the posterior cerebral artery produces hemianopsia, hemiplegia and sometimes hemianesthesia. That of the basilar artery produces clonic spasms, contracted pupils, spasm of muscles of deglutition and hemiplegia. In complete obstruction we find paralysis of both sides with symptoms of bulbar paralysis. Blocking of a vertebral artery produces symptoms of acute bulbar paralysis.

The unconsciousness which ushers in the attack is seldom as deep as coma and is of short duration. The paralysis is not complete and sometimes disappears within a few days, in other cases it persists through life. In the thrombotic form there are occasional mild apoplectiform attacks, each attack leaving the patient worse than before. Mental impairment is marked and in some cases there is a rapid progressive dementia. The aphasia is usually permanent. Mental and physical symptoms exhibit at times marked variations, the mind being sometimes quite clear while within a few hours there would be mental confusion with loss of memory. In the same manner the paralysis, aphasia, coördinating power, etc., may change within a few hours.

Diagnosis.—The differential diagnosis between cerebral embolus and thrombus depends upon the advent, the former being sudden, the latter gradual and generally with a preceding history of cerebral arteriosclerosis. In apoplexy the face is congested, the coma is complete and lasts longer than the unconsciousness of embolism, there is stertor, and the pulse is full and slow. In embolism there is generally a history of rheumatism or endocarditis and it comes on earlier in life than apoplexy.

Tumor of the brain may produce symptoms resembling thrombus. There are, however, no previous symptoms of arteriosclerosis. The advent is very slow, headache is persistent and any slight excitement will aggravate the latter and may produce spasms or unconsciousness. The symptoms are those of cerebral compression, namely, epileptiform convulsions, choked disc, facial paralysis, localized pain, etc.

Cerebral abscess generally has a history of injury or disease of the middle ear with septic symptoms.

In normal senile degeneration there is a gradual failing of the mental powers but there is no history of unconsciousness, paralysis or aphasia.

Prognosis.—The prognosis of cerebral softening is bad. While life may be prolonged for years in mild cases, an embolus in a main artery may cause rapid and extensive degeneration and death in a few days. The same conditions which lead to the formation of one embolus or thrombus will lead to the

formation of others and several attacks usually destroy life. Mental impairment leads to dementia.

Treatment.—The treatment is unsatisfactory as the same treatment which in one case apparently helps may have the opposite effect in another. The first indication is to maintain the strength of the heart by means of rapidly acting cardiac stimulants, preferably the hypodermic use of camphor and ether. Ammonia inhalation should be tried during the coma. After the first shock is past and the patient emerges from the coma, the further treatment depends upon his condition. If there is much irritability and mental confusion he must be kept quiet by narcotics. Cerebral excitement must be avoided but the treatment sometimes advocated in cerebral embolus and thrombus, which is to treat the case as one of cerebral apoplexy, is wrong. In apoplexy there is extravasation of blood with cerebral hyperemia and compression and the measures employed are to diminish the flow of blood to the brain. In embolus and thrombus we have the opposite condition. There is cerebral anemia beyond the point of occlusion, there is no compression, the face is usually pale and the pupils are unaffected. The first symptoms of apoplexy, embolus and thrombus are due to shock and we must look after the heart. After this, however, recovery from thrombus depends upon the rapid establishment of collateral circulation and for this purpose a full supply of blood to the brain is necessary. In these cases powerful cardiac stimulants are indicated. We must be certain of our diagnosis, however, for if used in apoplexy they may produce fatal results. The later treatment is symptomatic, electricity, massage and passive motion to overcome the paralysis, and phosphorus for the mental impairment.

CEREBRAL HEMORRHAGE

The frequency of apoplexy in the fifth, sixth and seventh decades of life, its sudden attack and the profound impression upon the whole organism, mentally and physically, make it perhaps the most readily recognized disease of old age. The essential lesion is the rupture of a miliary aneurysm or of an artery at a point where it has been weakened by the atheromatous process. The break generally occurs in some part of

the circle of Willis although it may occur in any artery or arteriole of the brain.

Etiology.—Any cause that produces arterial degeneration will also act as a predisposing cause of apoplexy. The most prominent of these causes aside from senile involution are alcohol, lead, mercury, syphilis, nephritis, gout, anemia, leukemia, and purpura. The exciting cause is usually some sudden strain which increases the blood pressure, some intense mental excitement, shock, alcoholic stimulation or other cause that would produce cerebral hyperemia. In many cases a heavy meal preceded the attack.

Pathology.—The essential lesion in cerebral hemorrhage is a ruptured vessel, one or more miliary aneurysms or an atheromatous artery. The grave, often rapidly fatal, attacks that occur in senile cases are usually due to the latter cause. The clot from a miliary aneurysm is small, while from a larger vessel it may be the size of a hen's egg. The nerve fibers that are compressed become sclerosed and degenerated. If recovery occurs the clot is not reabsorbed but breaks down and contains fatty granules, pigment and broken-down brain matter.

Symptoms.—Premonitory symptoms are rare and occur only if the exciting cause prevails for a long time or if there are exciting causes not sufficiently pronounced to cause rupture. In such cases there are usually headache, vertigo, thick speech and tingling in one hand or foot. Occasionally there is some impairment of the special senses, a feeling of weight or heaviness and intense mental depression. There is generally a momentary prodromal stage with terror, vertigo, weakness and numbness on the affected side, the patient tries to drag himself to a seat or corner, then falls insensible in a heap. The coma is complete, the face is red or cyanosed, the breathing stertorous, the pulse strong, full and slow, the sphincters are paralyzed permitting evacuation of the bladder and intestines. Hemiplegia invariably results, in rare cases there is a spasm or convulsion. The severity of the disease is determined by the severity of the coma and the extent of the hemiplegia. In a mild attack there is stupor from which the patient can be momentarily roused and from which he awakes in a few hours, his mind confused with perhaps some aphasia but able to swallow. In this case the arm and leg may be completely paralyzed but the facial and hypo-

glossal nerves are but slightly affected. It is hardly necessary to take up the localizing symptoms which would determine the exact location of the rupture and extravasation of blood. If the hemorrhage is into the medulla the cranial nerves are affected and death from interference with respiration and heart action results. Hemorrhage into a lateral ventricle generally produces rigidity of the opposite side, convulsions and death. In mild cases the coma clears up after a few hours leaving some mental confusion, aphasia, headache, vertigo, occasionally some sensory impairment. The paralysis also frequently clears up slowly, but the affected parts rarely fully regain their power. In severe cases it may take two or three days before the mind is sufficiently clear to respond to questions and even then there may be so much mental confusion that the patient cannot answer intelligently. Repeated gaping is an indication that the patient is passing out of the coma. After the patient passes into a stage of stupor he can be roused, but immediately relapses into the soporous state, which disappears slowly and weeks may elapse before he has regained his intelligence. Complete recovery of either intelligence or power is rare. More often there remains a postapoplectic dementia in which there is mental confusion with depression, occasionally fears and phobias, a dissatisfaction with the surroundings and rage at his impotent helplessness. With increasing mental weakness the patient becomes apathetic, his interest in the external world becomes less, but the dementia does not become complete and there is usually more intelligence than the dull, expressionless countenance would indicate.

There are numerous minor symptoms depending upon the extent and location of the extravasation but these do not affect the diagnosis or treatment.

Prognosis.—The prognosis is generally bad, especially if the cranial nerves are involved. A second attack is almost invariably fatal. The principal source of danger is in hypostatic congestion of the lungs and pulmonary edema. Absolute rest is necessary to prevent a second attack or further extravasation of blood and this rest favors pulmonary stasis. Rapidly forming bed-sores are very unfavorable signs and a rise in temperature during the coma is also unfavorable. A coma lasting over twenty-four hours is usually fatal and likewise the appearance of Cheyne-Stokes respiration. A favorable diagnosis can be

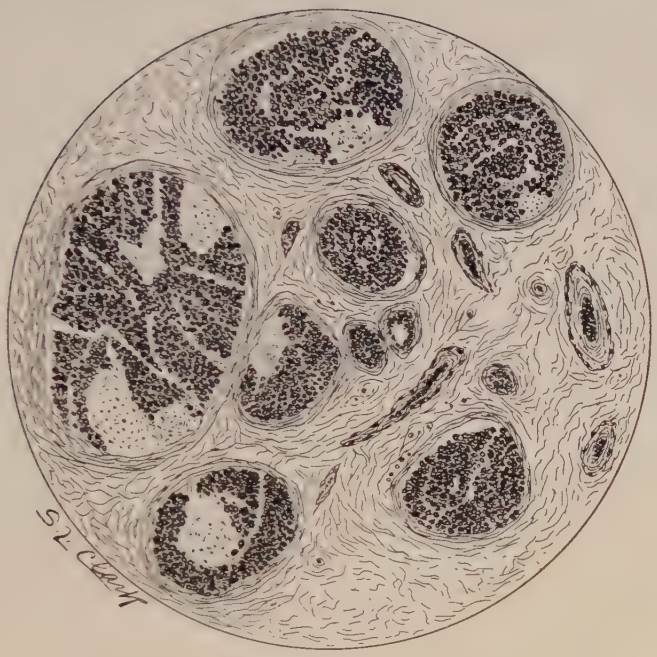
given if the coma clears up in a few hours after the attack, and if the fall in temperature did not exceed 2 degrees and if it does not rise above 103. A hemiplegia that does not improve in three or four months will never improve.

Diagnosis.—The only diseases which might be mistaken for apoplexy are cerebral embolus and thrombus. Cerebral embolus occurs at an earlier age, there is generally a history of rheumatism or endocarditis with valvular defect, or there may be a history of infarcts in other organs. The attack is not as severe, the coma not as profound, the face is pale and there are generally clonic spasms. In cerebral thrombus the symptoms come on more slowly and are not as severe, the face is pale, there are no spasms, the symptoms are altogether milder and may clear up completely. There is neither fever nor stertor in cerebral embolus or thrombus. In alcohol narcosis there is the odor of alcohol, and generally delirium and restlessness; there is no inequality of the pupils nor evidences of hemiplegia. The pulse is as in apoplexy. We must remember that apoplexy frequently occurs after a debauch and there may be the odor of liquor in addition to the signs of apoplexy. In this case we may find the contracted pupils of alcoholism but there is also evidence of paralysis, the paralyzed limb dropping more limply than the other, the latter responding to irritation. Diabetic and uremic comas give a causative history, the diabetic coma is usually preceded by dyspnea or vomiting and there is no paralysis, while in uremic coma if there is paralysis there are generally convulsions preceding the coma or during its progress. Examination of the urine will clear up a questionable diagnosis. Both forms of coma begin in stupor and proceed to complete coma. Other causes of coma as epilepsy, opium poisoning, cerebral concussion and compression, are readily distinguished from the coma of apoplexy by the history and pathognomonic signs.

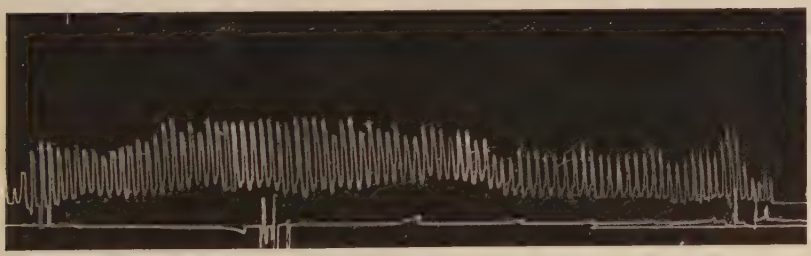
Treatment.—There is no routine treatment for apoplexy as the same method that will avail in one case will harm another. Venesection, highly praised by many authorities, is extremely dangerous in old age. It may be of service in robust, middle-aged individuals where there is a full bounding pulse and flushed face and the certainty of correct diagnosis. In aged persons the withdrawal of 10 or 15 ounces of blood may produce speedy collapse and death.

The most important rule in the treatment of apoplexy applies to the first few minutes after the attack. Raise the patient's head but *do not move him*. More harm is done by moving the patient upstairs or to a hospital within a few minutes after the stroke than by any subsequent treatment. He should not be moved for at least half an hour and in the meantime ice should be applied to the head and hot cloths to the feet, the object being to withdraw blood from the head by producing a local hyperemia in the lower extremities. Involuntary evacuation of the bladder and bowels generally occurs during the coma. If this does not occur the catheter and an enema should be used. During the comatose state drugs given by the mouth are liable to enter the larynx and bronchi and absorption by the stomach is slow or entirely inhibited. Whatever drugs are given during this time should be administered hypodermically. In case of threatened collapse camphor dissolved in ether should be given. Strychnine and digitalin can also be used but nitroglycerin is contraindicated. If there is a rapid full pulse aconite or veratrin should be used every half hour until the pulse slows down and remains slow. A rapid weak pulse indicates weak heart and threatened collapse. The most critical period is during the comatose stage and all our efforts must be directed to maintain the strength of the heart without increasing the cerebral hemorrhage. After the comatose stage has passed and the patient is able to swallow the further treatment is symptomatic. The one main precaution is the avoidance of anything that might produce cerebral hyperemia or increased blood pressure. After the comatose stage the patient should be occasionally moved to prevent hypostatic congestion and an air or water cushion should be provided to prevent bed-sores. If there is much restlessness morphine and bromides are indicated. For insomnia, veronal is best. Phosphorus is of service if there is dementia. For headache, frequently a distressing after-effect, cold applications to the head can be tried.

If the facial paralysis subsides within a week we can expect a subsidence of the hemiplegia. If it persists we have a difficult problem to deal with. Under no circumstances should any attempt be made to massage or institute other treatment of the affected limbs within a week after the attack, and even then only the mildest passive motion should be attempted. There



Chronic Interstitial Neuritis, Showing Degeneration in Some of the Nerve-fibers. (From Gordon's "Nervous Diseases.") The interstitial tissue is everywhere increased and the perineurium thickened. The patient had arteriosclerosis.



Tremorgraph—Post-hemiplegic tremor. (Neustaedter, *Med. Record*, July 17, 1909.)

is always the danger that rigidity and contracture of muscles will develop if the paralysis persists, and we are sorely tempted to prevent this by motion, massage, electricity or other means, but if attempted too early there is the greater danger of a second attack. After the second or third week we can begin with more active treatment, using vibrators, the faradic current and massage but voluntary exercise should not be permitted for several weeks, and the patient should be constantly cautioned against attempting to walk or using the arms until a fair amount of power has returned. During the first week concentrated liquid foods, preferably predigested or partly digested, should be used, but alcohol must be avoided and this eliminates most of the prepared liquid foods on the market.

SENILE NEURITIS

Senile neuritis is a form of chronic neuritis in which the senile changes in the nerves appear to be an etiological factor. It may occur as a localized or general neuritis.

Etiology.—In many cases an exciting cause can be found. This may be traumatism, sudden temperature changes, long exposure to cold, extension of an inflammation from adjoining parts, diabetes, alcoholism, lead or mercury poisoning, syphilis or other toxemias. Where there is a traumatic cause, the injury may be no more than a scratch, prick or bruise. The neuritis may then appear days or weeks after the injury. Decubitus is often preceded by neuritis. There are many cases in which no exciting cause can be found and aside from a concomitant arteriosclerosis the only assignable cause is the senile change in the nerve.

Pathology.—In some cases no structural change can be found; in others we find the changes observed in the ordinary interstitial and parenchymatous types of neuritis. The senile form of neuritis is generally a polyneuritis of the parenchymatous type, the changes being more marked at the periphery. The axis cylinder is apparently not affected but there is a hyperplasia of the neuroglia. The muscle supplied by the affected nerve undergoes fatty degeneration and the vessels become sclerosed.

Symptoms.—Cases of localized senile neuritis are rare and

the early symptoms are mild. There is never the intense pain associated with this type of neuritis in earlier life or when due to other causes, though the pain is constant and increased upon pressure. The reflexes are diminished but coordination is not affected unless the cord is involved. In some cases there is no marked motor or sensory impairment and the disease is discovered accidentally when pressing over an affected nerve and tenderness is found. In the multiple form of senile neuritis there are motor and sensory disturbances in several nerves, generally in those of the extremities, motion is diminished but there is never complete paralysis as occurs in traumatic neuritis, paresthesia especially pruritus occurs but there is little or no pain except upon pressure. The reflexes are diminished, the patellar reflex being usually lost, but there are no ataxic symptoms. In some cases the motor, in other cases the sensory manifestations predominate. Twitching and tremors are rare and muscle atrophy is a late occurrence. The disease being due to the progressive senile degeneration of the nerve, is progressive, but the symptoms can often be ameliorated.

Treatment.—If the pain is severe, a hot pack or some local anesthetic like cocaine, chloroform or a mixture of chloral and camphor will give temporary relief. The ethyl chloride spray should not be used, as the intense cold produced may destroy the surface capillaries. The treatment of the functional impairment depends upon the character of this impairment. If there is irritability, hot baths and internally large doses of bromide of sodium or potassium should be used. Diminished functional activity requires stimulation. Locally galvanism, vibration and massage can be employed. Internally strychnine and arsenic should be given, care being taken not to overstimulate the heart and to guard against the cumulative effects of the arsenic. When the strychnine is discontinued, caffein or theobromin can be substituted.

SENILE TRIFACIAL NEURALGIA

Trifacial neuralgia involving the terminal fibers of the third branch in the bony structure of the lower maxilla is the only form of neuralgia bearing a distinct relation to the senile processes.

Etiology.—This form of neuralgia affects the alveolar process of the toothless lower jaw and is probably due to compression of the terminal fibers in the bony structure. It is a compression neuritis rather than a true neuralgia, but the symptoms are those of the latter. A paroxysm is produced when an attempt is made to crush a hard substance between the jaws, or when the jaws are forcibly closed; even simple pressure upon the jaw or the presence of a cold substance as ice may bring it on. An attack may, however, come on without pressure or cold or any other discernible cause and whether due to pressure or any other cause the attack is identical in character with an ordinary neuralgic paroxysm.

Neuralgia in the other branches of the trifacial nerve as well as in other nerves may be due to an impoverished condition of the nerve caused by arteriosclerosis. It is, however, difficult to determine the basic etiological factor from the host of possible causative factors that are generally present. In some cases there is no structural change in the vessels or nerve and no other discoverable cause.

Symptoms.—The principal symptom of this form of trifacial neuralgia is a paroxysmal pain in the lower jaw, usually localized, occasionally occurring in several spots and if brought on by pressure the pain may be some distance away from the spot pressed upon. It may be a momentary stitch or a lancinating pain lasting from a few seconds to many minutes with complete remission in the intervals. There are usually several pressure points along the ridge of the alveolar process, besides the usual one at the orifice of the inferior maxillary canal which, when pressed upon, intensify the pain. In the intervals between the attacks mild pressure upon these points does not produce pain, but when a certain degree of pressure is reached it is immediately followed by the agonizing pain which marks the disease.

Treatment.—In the treatment of this form of neuralgia we must try to discover the basic etiological factor. If we find that it is a pressure neuritis and is brought on only by pressure upon the gums, the indication is plain; soft foods requiring no chewing must be given and hard particles must be avoided or crushed before being eaten. In many cases the neuralgic attacks come on when pressure upon the gums is excluded and we can

find no other cause. In these cases the treatment is purely empirical. If quick action becomes imperative a hypodermic injection of morphia and atropia should be given. A 2 per cent. cocaine ointment made with an animal fat base will generally give relief. A bit of cotton soaked in ether placed over the pressure point is also effective but the ethyl chloride spray which is serviceable in other localities cannot be used in the mouth. Whatever local treatment is used must be applied to the gums and this excludes many drugs which can be used upon the skin. The aconitin treatment which is almost a specific in functional neuralgias is generally inadmissible in senile cases on account of its depressant effect upon the heart and lungs. The combination of aconitin and digitalin, which has been recommended by some authors, is irrational, as the digitalin which is added to overcome the depressant effect of the aconitin is slow in action, while aconitin acts rapidly, and may cause delirium cordis and paralysis of the respiratory muscles before the digitalin has begun to act. If the heart is in good condition the aconitin may be given in doses of 1/300 to 1/200 grain combined with twice the quantity of atropia. Electricity in various forms, Roentgen therapy, light therapy, hydrotherapy, massage, vibrations and other non-medicinal measures have been tried but none give the uniform results obtained from aconitin. The injection of alcohol has given relief in some cases but it is intensely painful and the relief is but temporary. Surgical treatment is rarely indicated in this form of neuralgia. When all other measures fail and surgical intervention becomes necessary the best results are obtained from the removal of bone around the foramen by means of the galvanocautery or by resection and scooping out with a bone curette—Jaire's operation.

MODIFIED DISEASES OF OLD AGE

The diseases of the third group are not senile diseases, but diseases which may occur in earlier life. When occurring in the aged they differ so greatly from those of maturity that they can be differentiated into two separate diseases. The senile pneumonia differs in symptoms, prognosis and treatment from bronchopneumonia, for which it is frequently mistaken. Senile

cystitis differs from the cystitis of earlier life in etiology, pathology and treatment. This group could be made to include every disease occurring at both periods of life for the reason that the senile degenerative changes modify pathological processes, complicate symptoms, render prognosis more unfavorable and demand different treatment. When these differences are clearly marked, especially when a disease presents symptoms that are never found in maturity, it is placed in the third group. The prefix senile is added to these diseases and to all diseases in which it is necessary to differentiate between them and similar diseases occurring in maturity, such as senile gangrene, senile neuritis, etc.

HAY FEVER

When occurring in the aged this disease is almost always carried over from earlier life. It differs in some of its cardinal symptoms from the disease in maturity. Owing to the increasing atrophy of the mucous membrane, the coryza becomes milder year after year and may disappear entirely. There is usually an absence of conjunctivitis. There is, however, a dry irritating bronchial catarrh and asthmatic attacks with expiratory dyspnea, coming on at night especially if there is much moisture in the atmosphere. The bronchial symptoms increase as the nasal symptoms decrease. There is rarely fever or any other of the usual concomitants of the infectious diseases. (In this as in other diseases of this group, the symptoms that do not differ from the symptoms appearing in maturity, are generally omitted.)

The **treatment** of hay fever is prophylactic and symptomatic. The prophylactic treatment is the same as in maturity and consists of change of climate. Those who are attacked while in the lowlands or near the seashore will obtain relief in the hills or inland where there is a dry atmosphere. The reverse holds good for those who are affected when in the highlands or inland. Every sufferer must determine for himself the locality where he is free from attack. Persons who had remained away for years from the locality in which they were formerly attacked will be attacked again when they return to that place. Adrenalin and cocaine, virtually specifics in maturity, are useless and dangerous in old age. The asthmatic attack can be relieved by chloroform

cautiously inhaled and the internal administration of heroin in 1/10-grain doses.

SENILE ASTHMA

Etiology.—The term asthma is a diagnostic placebo which tells the physician no more than it does the patient; *i.e.*, that there is a spasmodic dyspnea. It is usually applied to bronchial asthma, a disease which is rare in the aged, although dyspnea occurs frequently at that time of life. Spasmodic attacks may occur in emphysema, cardiac disease, aortic aneurysm, dyspepsia, nephritis, diabetes and various nervous conditions of the aged.

Symptoms.—The forms included under the term senile asthma are pulmonary and cardiac asthma, these being directly due to the senile processes, emphysema and cardiac disease or arteriosclerosis of the coronary artery. Other forms of asthma and spasmodic dyspnea are readily distinguished from senile asthma. In *bronchial asthma*, which is rare in the aged, there is generally a history of attacks going back to maturity, there are the Curschmann's spirals, Charcot-Leyden's crystals, there are usually premonitory symptoms of sneezing, a tickling in the throat extending to the chest, causing a sense of irritation and coughing. In *dyspeptic* or *gastric asthma* the Curschmann's spirals and Charcot-Leyden's crystals are absent, the attack comes on after a heavy meal and there is inspiratory and expiratory dyspnea. The dyspnea of *aortic aneurysm* is inspiratory and expiratory, being due to pressure upon the trachea or bronchus, and it changes with change of position. Other symptoms pointing to aneurysm determine the diagnosis. *Hysterical asthma* is rare in the aged and there are other symptoms pointing to hysteria, while the breathing is slow or irregular. Spasmodic attacks of dyspnea may occur in nephritis, diabetes and various nervous diseases either through the effect of the disease upon the heart and lungs or upon the nervous regulation of these organs.

In senile asthma the distinctive spirals and crystals in the sputum are absent and there are evidences of emphysema or cardiac disease, usually both. In the emphysematous form there is the reversal of rhythm and difficulty in expiration, while these may be absent or irregular in cardiac asthma. The dyspnea of emphysema comes on after exercise; in cardiac asthma the attack may come on while at rest and it is accompan-

ied by palpitation or arrhythmia. This arrhythmia may, however, occur in emphysematous asthma, as it follows exercise and the heart is generally affected at that period. Cardiac asthma occurs occasionally at night, the patient awakening out of a nightmare with a choking sensation, palpitation and feeling that he must die from suffocation. The dyspnea in senile asthma gradually becomes less severe and finally passes away entirely, generally without cough or expectoration.

The **treatment** of senile asthma depends upon the cause. In the emphysematous asthma immediate cessation of exercise and lying down with the head raised will usually give relief. If the dyspnea persists it may be necessary to give a hypodermic injection of morphine 1/8 grain and atropine 1/120 grain. In cardiac asthma the cardiac disease upon which it depends must be treated. During a paroxysm the inhalation of a nitrite of amyl pearl will usually give immediate relief, contraindicated, however, if associated with chronic bronchitis. The inhalation of powdered stramonium, lobelia and other antispasmodics are useless and dangerous in either of these conditions. They are useful to relieve the paroxysm of bronchial asthma if there is no cardiac impairment. Many remedies which are useful in bronchial asthma in earlier life cannot be given to the aged on account of their depressing effect upon the heart. Chloral, antipirin, pilocarpine, are therefore contraindicated. Bronchial asthma usually lessens in severity with advancing age and gives way to the senile forms. If it persists and emphysema and cardiac disease can be eliminated, the iodides, preferably the syrup of hydriodic acid and bromides in large doses should be used.

PLEURISY

The pleurisy of old age differs in some features from the pleurisy of early life.

Etiology.—Senile pleurisy is almost always a secondary disease though the primary affection is sometimes so mild as to give no clearly defined symptoms. In some cases the primary disease is latent until the pleurisy is recognized and it then appears in an active form, being apparently secondary to the pleurisy. It most frequently follows pneumonia, especially when the inflammatory process lies close to the surface. A

septic infection, either local or general, may be followed by pleurisy, and pulmonary tuberculosis in the aged generally affects the pleura as well. It may also occur in typhoid fever and other infectious diseases. Tumors and inflammation in adjacent tissues may produce inflammation of the pleuræ, though these cases are comparatively rare. In some cases no cause can be found and the exudate is sterile. The disease is much milder than in maturity.

Pathology.—Pleurisy begins with a fibrinous exudate upon the inflamed site followed by an exudation of serum. The serum generally contains pneumococci, streptococci and staphylococci, occasionally tubercle bacilli, colon bacilli, etc. The septic organisms convert the clear serum into the thick, cloudy, purulent fluid found in empyema. In rare cases, there is a hemorrhagic exudate following tuberculosis or carcinoma. It may occur also as the result of traumatism or follow such rare causes as the hemorrhagic diathesis, pulmonary gangrene, bursting of an aneurysm, etc. The pseudochylous or chyloid exudate occasionally found after tapping is due to fatty degeneration of pus cells.

Much stress has been laid in recent years upon cytology and cytodiagnosis of pleuritic effusions. These laboratory adjuncts to diagnosis are rarely required except to furnish corroborative evidence of clinical and bacteriological findings. In the cytological examination the number and character of the polynuclear leucocytes, lymphocytes, and endothelial cells are considered. In pneumococcic pleurisy there is a polynucleosis, marked autolysis, the endothelial cells are numerous at first and later diminished in number. Streptococcic pleurisy has a polynucleosis, the cells degenerated and a large number of endothelial cells. Tubercular pleurisy has at first a lymphocytosis, numerous eosinophile cells and few red cells; later there is a polynucleosis, endothelial cells few and scattered. Typhoid pleurisy has a lymphocytosis and endotheliosis, increased eosinophiles and many red cells. In cardiac pleurisy there is a marked endotheliosis and sometimes a polynucleosis. In malignant pleurisy portions of the tumor mass are sometimes found. These cytological findings are often valueless, as there are usually pneumococci and streptococci present and the cytological changes produced by both are found.

The anatomical changes in the pleura itself due to pleurisy are not well marked. The senile pleura is normally thickened and the surfaces may be adherent without having given any clinical evidences of an inflammatory process. In senile pleurisy the surface may be reddened and it appears swollen and rough. There are frequently adhesions but it cannot be stated with any certainty whether they are the result of the pleurisy or are old processes. There is never an extensive exudate and the small amount of this with the tendency to become fibrinous would favor the formation of adhesive bands and surface adhesions.

Symptoms.—Acute idiopathic pleurisy is rare in the aged and the classical onset of this condition is seldom observed. The slight chills which usher in acute pleurisy in earlier life are usually absent. There may be fever due to the primary disease, but the senile pleurisy itself causes but slight if any elevation of temperature. In some cases where puncture revealed an empyema there was no fever. The pain is usually slight. There is occasionally a sharp, momentary pain or "stitch" over the site of the inflammation, more often there is a dull ache which becomes a sharp pain only when coughing, or taking a forced deep breath. If the diaphragmatic pleura is involved, the pain may radiate into the abdominal cavity and simulate intestinal colic or peritonitis. Dyspnea occurs frequently owing to the compression of the lung by the exudate. This may also produce cyanosis especially after exercise. The exudate may also press upon the heart interfering with its motion, causing arrhythmia or palpitation and may produce such cardiac disturbances as to cause death. The respiration is shallow, the patient being afraid to take a deep breath on account of the pain. During the early stage of pleurisy, before the serous exudate has developed, there is a dry painful cough without expectoration. This ceases as soon as the exudate keeps the inflamed surfaces apart. If there is a cough with expectoration it is due either to bronchitis or pneumonia. An empyema may, however, open into lung tissue and cause a purulent expectoration. The patient will bend toward the affected side and when lying down before the serous exudate has appeared, he will lie on the unaffected side so as not to compress the inflamed pleura. After exudation he will lie on the affected side, so as to allow full expansion of the clear side of the chest.

The physical signs are not so clearly defined as in maturity. Owing to the rigid chest walls which move as a whole, no change in movement can be noted on the two sides but there is usually a bulging in the intercostal spaces of the unaffected side. As there is generally a senile emphysema with chest rigidity the percussion and auscultation sounds are altered. The percussion note is dull rather than flat and the note on the healthy side is more tympanitic than usual. The affected side lies lower in the chest than the other and may displace the abdominal viscera below it. The auscultatory sounds are not lost entirely as in maturity. Friction sounds before the exudate are quite clear and after the serous exudate, the vesicular breathing becomes faint. Bronchophony is found above the level of the fluid. The percussion and auscultation sounds are altered when the level of the fluid is changed by change of position. When the exudation is reabsorbed or removed the normal sounds are heard again, although pleuritic friction sounds may be heard for months after complete recovery. The X-ray can be employed in corroborating the diagnosis. The symptoms of pleurisy are modified and may be partly or completely masked by the symptoms of the primary disease. The diagnosis is especially difficult if there is a co-existing pneumonia localized in the lower part of the lungs. In this case the early symptoms are often completely masked by the symptoms of the pneumonia. In some of these cases the first indication of pleurisy is the finding of friction sounds or a pain on coughing. More often there will be no early signs or symptoms, and not until the serous exudate hides the râles of pneumonia is the presence of pleurisy suspected. In many cases the only positive means of diagnosis of pleurisy is an exploratory puncture or aspiration. Empyema will generally give an irregular elevation of temperature, but even this may be absent in the aged, especially if there is a complicating pneumonia. Baccelli's test, a whisper heard clearly in serous pleurisy and lost in hemorrhagic and purulent pleurisy, is fairly accurate, nevertheless, the only certain test is the exudate obtained by puncture.

Treatment.—The treatment is mainly symptomatic plus the treatment of the primary disease. The special indications are the relief of pain, the removal of the fluid by withdrawal through aspiration or resorption, reduction of temperature and

sustaining the strength of the patient. As the disease is usually mild in the aged, it is rarely necessary to do anything for the pain or temperature. If the pain is severe, local applications, such as belladonna plaster or belladonna ointment, cocaine ointment, dry or moist heat, turpentine stupes, dry cups, etc., will usually give relief. Narcotics and analgesics are seldom required.

As the rise in temperature is usually due to the primary disease except when empyema develops, the measures applicable in the primary condition are indicated. Quinine is the best antipyretic in septic conditions in the aged. Cold baths are dangerous on account of the shock and deficient reaction and the coal tar products are cardiac depressants.

The removal of the exudate is the principal indication. The most certain measure is aspiration but there are several dangers connected with this procedure in the aged. The dread of anticipation followed by the pain of the momentary puncture will produce a shock causing an unfavorable reaction. The rapid removal of the fluid produces a partial vacuum, and to fill this the lungs must expand rapidly. As the emphysematous lung had been compressed by the fluid, its rapid distention causes distention of the alveoli with rupture of their walls and an increase in the emphysema. The puncture site is frequently the focus for a septic or an erysipelatous infection. Aspiration should be performed only if other measures fail and the pressure of the effusion upon the heart or large vessels causes serious interference with the circulation or when the pressure upon the lungs causes cyanosis or distressing dyspnea. The puncture is made in the eighth or ninth intercostal space on the scapular line. A small quantity of serum should be withdrawn slowly and the opening rapidly closed. In some cases resorption is brought about through the local application of cataplasms, or tincure of iodine, and the internal administration of iodide of potassium and salicylate of soda in 5-grain doses of each. This should be tried in every case after the evidences of serous effusion appear. In empyema surgical measures are necessary. Serum therapy has been fruitless in these cases, resorption is impossible and it is impossible to withdraw the pus completely by puncture and aspiration.

PULMONARY HYPEREMIA

Pulmonary hyperemia may be active or passive, local or general. The form usually found in the aged is passive hyperemia or pulmonary congestion. *Active pulmonary hyperemia* is rare in old age. It is generally due to direct irritation of the lung through the inhalation of irritating vapors or gases, cold air, rarified or compressed air, violent coughing, rapid heart action, excessive exercise, etc., or it may occur as a *compensatory hyperemia* in a healthy part of the lung when the circulation is impaired in another part, as happens in pneumonia. Infectious diseases will sometimes cause active hyperemia.

Etiology.—*Passive pulmonary hyperemia* is generally due to diminished aspiratory energy of the left heart, or to mitral disease causing obstruction to the return circulation. These causes will produce a general congestion. If the patient is laid up for any length of time, maintaining a recumbent position, especially on his back, the blood gravitates to the most dependent portion of the lung causing a hypostatic congestion. The usual site of hypostatic congestion is the lower posterior portion of the lung.

Pathology.—Virchow first described the congestion due to mitral disease. In this form the lung is heavy, firm, of a dark red color dotted with yellowish or brownish spots. The alveolar walls are thickened and contain pigment granules while the alveoli themselves contain loose degenerated and pigmented epithelium cells. The larger blood-vessels are enlarged and engorged, while the capillaries are greatly dilated and lengthened and some may be ruptured, the blood extravasating into the walls and interior of the alveoli. A brownish fluid exudes from the cut surface. This form is called brown or pigment induration. In hypostatic congestion the lung is very dark and heavy, with little crepitation, the tissue is friable, the aveolar walls are swollen, the alveoli filled with degenerated cells and the vessels are tortuous, dilated and engorged.

A form of passive hyperemia occurring with some of the infectious diseases is splenization, the pathological changes causing a resemblance to spleen tissue. The lung is dark brown or purplish, while the cut surface presents many reddish or

yellowish spots where blood had extravasated into the tissues. The alveolar walls are swollen but the alveoli may be collapsed or filled with débris of degenerated epithelial cells. There is an interstitial edema which causes an oozing of watery fluid from the cut surface. This form of congestion is rare in old age.

Symptoms.—The principal symptoms of pulmonary congestion are dyspnea, cough and expectoration. These symptoms may occur in other pulmonary diseases and it is sometimes impossible to make a definite diagnosis. Pulmonary congestion is frequently found upon autopsy, which gave no symptoms during life and in many cases the first symptoms of an existing congestion are the symptoms of a rapidly fatal pulmonary edema. In acute hyperemia the symptoms come on suddenly or rapidly, in passive hyperemia they come on slowly, the hypostatic congestion existing sometimes for weeks before the dyspnea or cough becomes sufficiently distressing to receive attention. In brown induration the disease progresses still more slowly. The cough is loose and produces little distress. The dyspnea is severe in splenization and hypostatic congestion, but in brown induration the process of the disease is so slow that the patient gradually accustoms himself to the progressive diminution of sufficiently oxygenated blood. Physical exercise naturally increases the dyspnea. There is no pain and in some cases there is no distress other than a feeling of tightness, or oppression in the chest. When the disease is far advanced and aeration becomes much impaired cyanosis appears and prostration occurs, followed by pulmonary edema and death. The expectoration in brown induration contains degenerated, pigmented alveolar cells but rarely blood. In the hypostatic form there are blood streaks and spots. The expectoration is profuse and watery, differing from the blood-streaked sputum of pneumonia which is scanty and tenaceous. The respiration is hurried and in hypostatic congestion it is confined to the upper part of the lungs.

The physical signs are dulness upon percussion over the affected area, moist râles and absence of the vesicular murmur. In hypostatic congestion prolonged change of position will shift the location of the congestion or cause it to disappear altogether, but it will return upon prolonged rest in the original position. The congestion in the brown induration is permanent.

The vesicular murmur in hypostatic congestion is feeble or lost over the affected area; in brown induration the inspiratory sound is rough, while expiration is prolonged. Râles appear with the edema and are really symptoms of pulmonary edema (see Pulmonary Edema). Although the symptoms and signs of pulmonary hyperemia may occur in other pulmonary diseases the diagnosis is not difficult. Absence of fever and slow progressive onset distinguish passive hyperemia from acute inflammatory conditions. The sputum alone will suffice to differentiate it from pneumonia. The early stage of chronic interstitial pneumonia gives similar symptoms but the history will serve to distinguish between the two diseases.

Treatment.—The treatment depends upon the cause. In hypostatic congestion frequent change of position is necessary. In all forms the underlying cardiac disease must be treated. Occasionally the Bier hyperemia treatment or other measure for producing local hyperemia in some other part of the body will break up a passive pulmonary hyperemia, expectorants like senega, ipecac, squills, are useful, but the narcotics are contraindicated. Compensatory hyperemia requires no treatment.

SENILE PNEUMONIA

Faulty nomenclature, multiplicity of terms, diverse views as to the etiology and pathogenesis of pulmonary inflammation, and as to the interpretation of its signs and symptoms are responsible for the confusion that exists relative to the various pathological conditions included under the term pneumonia. Some authorities will include under this term only those conditions that are due to bacterial activity, others include non-bacterial inflammations of the lung. A simple hyperplasia without inflammation is called interstitial pneumonia and many writers call capillary bronchitis, bronchopneumonia though the lung tissue may not be involved. All forms of pulmonary inflammation, infectious and non-infectious, may occur in old age and when occurring are modified by the senile changes. (As the infectious forms are really constitutional diseases with localized manifestations they will be treated under the infectious diseases of the fifth group.) The term senile pneumonia is here applied to a non-infectious

inflammatory process in the lung of the aged. It may be localized or diffuse, primary or secondary, acute or chronic, mild or virulent, or latent, masked or abortive. In many cases a non-infectious pneumonia becomes an infectious one, the diseased tissue being a fruitful field for germ growth and development.

The primary senile pneumonia is generally acute and in most cases rapidly fatal. The secondary form is usually latent or masked and its existence is frequently not suspected until the terminal edema sets in, or if death is due to another cause, its presence is first discovered upon autopsy. The acute form is generally virulent, the secondary form is usually mild. Both forms may be localized or diffuse, the extent depending mainly upon the cause. Several localized areas may, by extension and consolidation, become a single large area of inflammation.

Etiology.—*Primary senile pneumonia* is generally due to sudden temperature changes when the surface becomes chilled or cold air is inhaled. Loomis says nine-tenths of all cases occur between November and May. Owing to the weakened heat regulation in the aged, reaction to the sudden chilling of the surface is neither rapid not complete and the blood dammed back from the surface engorges the adjoining viscera. In many senile cases there is a pulmonary congestion due to valvular disease and the sudden influx of blood converts the passive hyperemia into an active inflammation. When cold air is inhaled an active hyperemia must be produced in order to raise the cold air to the temperature of the body. The usual effect of the breathing of cold air is a bronchitis produced by the frequent alternation of temperature of the inspired cold air and the expired warm air. The capillaries of the vesicles become alternately engorged and contracted and thus an inflammatory process is instituted. Noxious and irritating vapors cause inflammation by irritating the lining of the bronchi and vesicles.

Secondary senile pneumonia generally follows a hypostatic congestion or brown induration, the passive hyperemia being converted into a low inflammatory process, through blood changes occasioned by another disease. It may also occur through the extension of a bronchitis or pleurisy, rarely a pericarditis.

Pathology.—The usual pathological changes are such as occur in pulmonary hyperemia, followed by red hepatization

of a primary pneumonia. The lung usually presents several small or one or two large areas of surface hyperemia and upon section there may be a uniform dark brown, smooth, moist surface, or if the disease is diffused there will be dark brown spots or patches. Under the microscope the alveoli appear larger, due to the emphysema which is almost invariably present, and the vesicles are filled with the débris of degenerated epithelial cells, mucus, blood cells or blood pigment and sometimes fibrin. The capillaries are enlarged and engorged and some may be ruptured. There is sometimes an interstitial edema and it is often difficult to determine whether there is an inflammatory process or a purely mechanical hyperemia with edema. In senile cases presenting no pulmonary symptoms until the terminal edema, it is generally impossible to differentiate between senile pneumonia and hyperemia with edema. The presence of fibrin in the alveoli, found at autopsy, always indicates an inflammatory process.

Symptoms.—Senile pneumonia generally begins with symptoms so mild that they are not noticed until the disease is far advanced. A distinct chill and fever indicate infection. In primary senile pneumonia there may be a sensation of chilliness after exposure, the patient feeling that he cannot get warm, that he feels cold all through. This is followed by malaise, languor, and uneasiness, the patient says he does not feel well yet cannot assign the discomfort to any locality. He may say he has not slept well and is disinclined to leave the bed. After a day or two of such vague sensations there may be a slight fever, headache, dyspnea, the breathing becomes shallow and rapid, a cough with little mucous expectoration follows, or an existing bronchitis is aggravated. There is no pain but an oppressive feeling in the chest, the patient frequently striking his chest as though to dislodge some mucus. He becomes rapidly weaker and dies from exhaustion. In other cases cerebral symptoms appear after the second or third day and the patient has a low muttering delirium which the family mistakes for talking in his sleep. In some cases pulmonary edema sets in; this occurs generally in secondary senile pneumonia following another disease (not secondary through extension of an adjoining inflammation but through blood changes). In another set of cases the disease begins with rapid prostration followed by

the pulmonary symptons of rapid, shallow breathing, dyspnea, a distressing cough and a tenacious mucous expectoration. A pneumonia following a bronchitis, pleurisy or pericarditis begins with a marked rise in temperature, while if secondary to a disease producing blood changes the symptoms may be latent or masked by the more pronounced symptoms of the primary disease. The symptoms common to all forms of senile pneumonia are prostration, rapid shallow breathing and cough. The dyspnea may not be pronounced, as in his exhausted condition the patient will not make the powerful efforts to get air and there will be cyanosis instead. Striking the chest apparently to dislodge mucus occurs generally when the air vesicles become filled with mucus or other material. The expectoration may be blood-streaked when a powerful effort is made to bring up the mucus and a capillary is ruptured by the strain of coughing. While primary senile pneumonia, occurring when the patient was healthy, gives early symptoms of pulmonary disorder, secondary senile pneumonia may give no clear symptoms at any time. An increase in temperature during an acute disease points to some complication; an increase in the rate of respiration during the course of an acute or chronic disease points to pneumonia. The respiration is usually so shallow that, unless there is an accompanying dyspnea, attention is not attracted to it. The neglect to count the respirations is responsible for many errors in the diagnosis where pneumonia is present. In hypostatic pneumonia following hypostatic congestion, the lower portion of the lung is affected and the physical signs are found over the posterior lower portion of the lung. Where scattered areas are involved, the physical signs can often be found at the apex, in the interscapular space and in the infraclavicular spaces and occasionally at the sides. There is percussion dulness over the affected areas, adjoining tympanitic areas, and fine crepitant râles are heard at the end of inspiration and beginning of expiration. The expiration is prolonged. The respiratory sounds are diminished and vocal fremitus may be increased. The percussion note may be altered if there is a portion of emphysematous lung over the inflamed area, the fine râles may be masked by the coarser râles of bronchitis or by the friction sounds of pleurisy or pericarditis.

In differentiating between senile pneumonia and other con-

ditions we have no pathognomonic sign, no symptom complex to guide us and it is often necessary to make our diagnosis by exclusion. *Acute inflammations* can generally be excluded by the absence of fever, or initial chill. The *infectious pneumonias* can be excluded by the absence of high temperature and pathogenic germs in the sputum. *Tuberculosis* is slow, there is not the rapid prostration, it responds to the tuberculin test and the bacilli are found in the sputum. *Influenza* has marked initial symptoms, fever, mucous inflammation, conjunctivitis and presents pathogenic germs. *Pleurisy* with effusion gives percussion flatness with change of level when the position is changed, the intercostal spaces bulge, no respiratory sounds are heard through the effusion, the cough is distressing and there is not the feeling of irritating mucus which induces the pneumonic patient to strike his chest in an effort to dislodge it. The secondary pneumonia following pleurisy, is either a hypostatic pneumonia giving no symptoms until edema sets in, or an infection with rapid rise of temperature. *Capillary bronchitis* or bronchiolitis gives no percussion dulness, expiration is not prolonged, there is not the profound or rapid prostration and the râles may disappear for a time after expectoration. If pneumonia follows a bronchitis the temperature is suddenly raised, the cough is more severe, expectoration is scantier, the respiration is hurried and shallow, there may be dyspnea and cyanosis and rapid prostration. In all cases of secondary senile pneumonia a rapid increase in the gravity of the primary disease with increased temperature, prostration and shallow, rapid breathing indicates a pneumonic complication.

Latent pneumonia may exist as a primary disease without giving any symptoms and the patient expires suddenly while apparently in perfect health, or there may be vague symptoms of malaise and weakness lasting several days when pulmonary edema suddenly develops and is rapidly fatal. This form of pneumonia occurs frequently in senile dements whose weakened mental powers are unable to comprehend the urgent symptoms.

Inhalation, aspiration and *deglutition pneumonias*, generally classed as bronchopneumonia, are due to the inhalation of noxious or irritating vapors or to the lodgement of a foreign body in the trachea or bronchi. The symptoms of deglutition pneumonia are localized to the part in which the foreign body

is lodged and later to the part of the lung supplied by the branches of the bronchus which is blocked. The inhalation pneumonia is a diffuse pneumonia from the onset. These forms of pneumonia are rare in the aged and when occurring give pronounced symptoms of primary senile pneumonia with intense dyspnea and rapid prostration. They begin as a non-infectious inflammation but infection soon sets in.

Treatment.—The principal indication in the treatment of senile pneumonia is the prevention of the fatal complications of general exhaustion, cardiac exhaustion, hypostatic congestion and edema and cerebral complications. The most frequent cause of death is cardiac exhaustion and this is often traceable to the injudicious use of cardiac stimulants. When the heart is working near the limit of its functional capacity further stimulation will suddenly paralyze or rapidly exhaust that organ. Cardiac stimulants should never be given in pneumonia before the heart becomes weak. Cardiac therapy in pneumonia follows the general rule for cardiac therapy in other conditions. A full rapid pulse requires cardiac depressants like aconite, veratrum and gelsemium cautiously administered. The coal tar depressants should never be given. A weak rapid pulse requires heart tonics like digitalis, strophanthus, coffee or cactus. A weak slow pulse requires spartein or strychnine. A full slow pulse requires the nitrites. Emergency drugs in threatened heart failure are strychnine, ether, camphor, the combination of strychnine, nitroglycerin and digitalin used hypodermically and alcohol or ammonia carbonate internally. In senile pneumonia the heart is weak from the start and mild cardiac stimulants like caffein or cactin can be given. As soon as heart failure is threatened, the more powerful stimulants are required. Digitalis should not be given, as on account of its powerful vasoconstrictor effect, the blood supply to the lungs is still further diminished. The rapid prostration is best combated by concentrated food, alcohol, strychnine, arsenic and phosphorus. The strychnine and arsenic can be combined as arsenate of strychnine given in doses of 1/100 grain. Phosphorus should not be used until there is marked exhaustion when it can be given in doses of 1/100 grain of the ordinary yellow phosphorus or 2 grains of the amorphous red phosphorus.

Frequent percussion is necessary to determine the presence of hypostatic congestion. The treatment is repeated change of position.

Cerebral symptoms may be due to high temperature, toxemia, disturbed cerebral circulation or deficient oxygenation of blood, the last being the usual cause in senile pneumonia. High fever does not occur in senile pneumonia, and the only toxemias aside from pathogenic bacterial toxins that may occur are autointoxications from the absorption of the products of intestinal decomposition or from retention of urea. Carbonic oxide intoxication, due to incomplete aeration, is evidenced by cyanosis. Respiratory stimulants are required, the most powerful being atropine. It can be given in doses of 1/120 grain. If there is a sallow cyanosis beginning with a pale face, nitroglycerin hypodermically or nitrite of soda by mouth should be added to the atropine. If there is a purplish cyanosis beginning with a flushed face, the atropine should be given alone. Oxygen is of temporary utility in relieving the cyanosis and cerebral symptoms but it does not influence the inflammatory process. Urea intoxication is infrequent in senile pneumonia. The treatment of it consists in renal stimulation by vegetable diuretics. If rapid action is required, as in threatened uremia—a rare contingency in senile pneumonia—the nitrate of soda or potash should be used in 5-grain doses every three hours. For intestinal autointoxication the proper remedies are active cathartics and intestinal antiseptics, preferably salol and the sulphocarbolates. There is no specific treatment for senile pneumonia. In some cases a hyperemia produced in some other portion of the body will relieve the pulmonary congestion. This can be done by hot foot baths, hot cataplasms applied to the chest or back or dry cups. These measures may avail in the beginning of the disease but not later. In ordinary cases none of the symptoms, except perhaps the cough and difficult expectoration, are distressing enough to attract the attention of the patient. There is rarely any pain but there may be a feeling of tightness or oppression in the chest. This may sometimes be relieved by hot poultices. Narcotics, especially the opiates, should not be given in this disease. If there is a distressing cough with little expectoration we can use equal parts of the syrup of the hypophosphite of ammonium, syrup of senega and syrup of

ipecac in teaspoonful doses. The inhalation of a solution of menthol in eucalyptol will stimulate the secretion from the mucous membrane of the bronchial tubes. For the dyspnea we can use a combination of atropine and strychnine, using 1/120 grain of atropine and 1/120 grain of strychnine. It can be given by mouth or hypodermically. Dyspnea is, however, rarely severe enough to require treatment. For insomnia we can use veronal, proponal or urethane. The carbamide group of hypnotics is safer than the methane group where there is impaired aeration of blood.

Hygienic regulations are of the greatest importance in senile pneumonia. Fresh dry air, sunshine, a temperature between 70° and 75°, no draughts, are imperative. A cheerful companion has a beneficial effect. There should be a daily evacuation of the bowels and the quantity of urine and urea passed daily should be noted. During the height of the disease we can use the liquid predigested foods in addition to simple carbohydrates but no meat. If convalescence occurs the patient should be instructed to take deep breaths even if they do cause spells of coughing. Warm clothing must be worn for months after recovery.

SENILE ACUTE GASTRITIS

This is the disease known to the layman as acute indigestion and is a frequent cause of death in the aged. It is due to an irritation of the degenerated stomach, generally caused by some dietetic error.

Etiology.—We must remember that the senile stomach has atrophied walls, there is a waste of the glandular element, and usually dilatation, which is more pronounced in beer drinkers and in those who habitually take too much food. There is a chronic gastric catarrh with slow digestion and abnormal fermentation. In this condition, causes too slight to be of any deleterious effect in the normal stomach of maturity will have grave results in the aged. The most frequent cause is overeating, especially while the stomach is still partly filled with food which is in the process of active fermentation. This accounts for the many cases of acute gastritis during banquets when food is taken shortly after the regular meal. In some

cases the cause can be traced to the ingestion of partly decomposed food, especially cold storage, canned and chemically preserved food.

Pathology.—The mucous membrane of the stomach is reddened but there is never the intense congestion found in the same disease of maturity. Swelling of the membrane is rare but erosions are frequently observed. The secretion of mucus is diminished owing to the waste of the mucous glands. The presence of a large quantity of mucus in the vomitus indicates that some intense irritant to the glands has stimulated them to abnormal activity. The stomach is distended with food and gas, pressing upward when the patient is erect, and bulging outward when he is lying down.

Symptoms.—The earliest symptoms are usually a feeling of distress and heaviness in the stomach with eructation of gas. Sometimes the pressure upon the diaphragm produces singultus, more often it causes gastric asthma through interference with cardiac action, and we have then symptoms of cardiac irritation, palpitation of the heart, weak, irregular pulse, dyspnea, vertigo, faintness and pallor. If the face is flushed there is danger of apoplexy. Vomiting may occur, although it is rare in old age. The eructation of gas gives temporary relief and if vomiting occurs the relief is more permanent.

Treatment.—The greatest danger from this form of gastritis is cardiac irritation; secondary dangers arise from prostration and from the direct injury to the gastric walls. The first danger is temporarily removed by putting the patient upon his back, thereby relieving the pressure upon the heart from below After a few minutes the patient can be placed in a semirecumbent position and pressure can be made over the stomach to dislodge the gas. It is a mistake to prevent eructations. The patient attempts to suppress this, owing to his mistaken sense of propriety, but the physician who attempts to prevent it is guilty of the grossest ignorance. The rational treatment in these cases is to empty the stomach as soon as possible. Where the stomach is overfilled, emetics will have little effect unless given in such large doses as to injure the walls of the stomach. The senile stomach is less sensitive to gastric irritants and the emetic mixing with the mass of food either loses its action or the action is slowed and prolonged. The most rapid and reliable emesis in these

cases is produced by a hypodermic of 1/10 grain of apomorphine. This should be combined with 1/2 grain of caffeine if the heart is weak. If vomiting occurs while the patient is unconscious he should be placed upon his side or almost upon his face with the head lowered to prevent aspiration of vomited matter. The strain of vomiting may be severe enough to produce prostration. In this case we can give strong black coffee or champagne or some other alcoholic stimulant greatly diluted. In an emergency where there is danger of death from exhaustion we must resort to the more powerful drugs like strychnine, digitalin, camphor, etc., given hypodermically. A localized pain after an acute attack indicates an erosion which may develop into an ulcer or may be the focus for a carcinoma. If such pain exists the subnitrate of bismuth in 5-grain doses is indicated and if the pain is severe or there is persistent nausea we can add cocaine in 1/8-grain doses. If the gastric attack is mild, vomiting should be induced by irritating the fauces or by giving 15 or 20 minims of the fluid extract of ipecac, mustard, or salt water or any other handy emetic. The same measure should be taken if an acute attack is due to decomposing food. After some relief has been obtained a vegetable cathartic should be given combined with the bile salts. Occasional massage over the fundus may be necessary to secure dislodgment of food that may be lying in the pouch as the flaccid walls of the stomach might permit a sinking of the fundus below the pyloric orifice. No food should be taken for several hours after the stomach has been emptied.

Simple Chronic Gastritis

Etiology.—*Simple chronic gastritis* is generally due either to repeated attacks of acute gastritis or to the constant irritation of the stomach by improper food or drink. It may also occur in gastric ulcer or cancer, in diseases which cause local passive hyperemia, in diseases of the liver with impaired portal circulation, in heart disease, and in some constitutional diseases as diabetes, gout, anemia, tuberculosis, etc.

Pathology.—The walls are usually thickened, the mucous membrane is pale and covered with a thick layer of mucus. If there is passive hyperemia the veins become dilated and

are prominent. The glands are sacculated and may form cysts if their openings are blocked. Late in the disease, the walls grow thin, but shrinking of the organ which occurs frequently in maturity does not occur in the aged.

Symptoms.—The symptoms of chronic gastritis are the same as those of acute gastritis, but milder and more persistent. There is a constant sense of discomfort about the stomach more pronounced when food is taken. Morning nausea and retching occur and bile-stained mucus may be vomited. In some cases food is vomited after every meal and (if this occurs) several hours later the vomited material has a sour odor from acid fermentation. Constipation, intestinal colic, palpitation of the heart and psychic depression are usual secondary symptoms.

Treatment.—The treatment which is dietetic, hygienic and medicinal should follow the lines laid down for senile gastric catarrh; alcoholics should, however, be forbidden. If it is secondary to another disease the cure may depend upon the treatment of the primary disease.

SENILE DIARRHEA

Etiology.—Diarrhea occurring in the aged is generally due to the faulty character, too great frequency or excessive amount of food taken. Owing to the senile changes in the stomach and intestines with diminished metabolic activity, a smaller amount of food is assimilated. If the appetite is not diminished and as much food is taken as during maturity, the demands on the stomach and intestines become excessive and frequent stools result. In some cases food passes unchanged into the intestines and may pass out unchanged causing senile lientery. Diarrhea may occur as a symptom of inflammatory or ulcerative conditions of the bowel but these can be readily distinguished from the simple chronic diarrhea of the aged. (See Enteritis.)

Symptoms.—Senile diarrhea comes on slowly, the patient becoming gradually accustomed to two or three stools a day. If the amount of food is not diminished the number of stools or the quantity of the feces will be increased. The stools may be normal in color and consistency or they may be lighter and thinner than normal but they are never fluid. There is no pain or tenesmus and aside from the frequency of the stools there

is no discomfort. They contain little or no mucus and no blood.

Catarrhal diarrhea is infrequent in the aged as the mucous membrane is atrophied, and its secretion diminished and it requires a violent irritation to produce an enteritis with mucous discharges. In this form of diarrhea there is some mucus in or surrounding the stool mass and when due to an acute inflammation or ulceration there is usually blood, and occasionally pus.

Serous diarrhea, due to intestinal irritation from undigested food is a watery, brown, offensive discharge which irritates the anus, and if it passes through impacted feces, it breaks off portions which come away in scales.

Nervous diarrhea is due to strong emotion and stools first normal are followed by a watery discharge.

The diarrhea of *chronic intestinal catarrh* is a mucous diarrhea generally with pain and borborygmi. The stools are not frequent, often not exceeding one daily, and they are sometimes of normal appearance, sometimes watery. (See Enteritis.)

The diarrhea of *simple ulcerative colitis* contains particles of undigested food and occasionally blood, the diarrhea alternates with constipation and there is pain or an uncomfortable feeling in the lower part of the bowel, besides loss of strength, emaciation, cachexia, etc. It occurs most frequently in men past middle age.

In all ulcerative and inflammatory diarrheas the lessened intake of food will diminish the amount, but will not alter the character of the stool. Intestinal parasites may cause diarrhea but these generally give symptoms pointing to their presence. In all cases of diarrhea an examination of the stools is necessary to determine the presence or absence of blood, mucus, pus, undigested food, shreds of tissue, and parasites.

Treatment.—The first indication in the treatment of senile diarrhea is to regulate the food, diminishing the quantity and frequency. Food should not be given oftener than in five-hour intervals. If there is lientery, the character of the undigested food should be determined and if possible the digestant of that form should be supplied or that variety of food withdrawn. Light colored and offensive smelling stools require the bile salts. In some cases a senile diarrhea can be cured by giving a

saline cathartic followed by a day's starvation. This should be the routine treatment of every case and the further treatment regulated by the result. If the diarrhea persists bismuth subcarbonate should be given in 5-grain doses every two hours for a day, then every four hours until the stools are normal in quantity and frequency. It is rarely necessary to resort to the mineral astringents, lead sulphate or copper sulphate. Should it be necessary, either one can be given in 5-grain doses three times a day. A starch enema will check a profuse diarrhea. In every case the regulation of food is of primary importance.

SENILE CYSTITIS

Etiology.—This is a form of chronic cystitis due to distention of the bladder and the presence of decomposing urine retained either in the cystic pockets formed in the process of senile degeneration of the organ or in the bladder through prostatic obstruction. When bacteria are introduced from without, as by a dirty catheter, the disease begins as a mild acute cystitis. In many cases of chronic cystitis there is a history of gout but the connection between the two is uncertain. The old theory that the enforced rest imposed by an attack of acute gout causes retention of urine with atony and dilatation of the bladder, the retained urine producing a chronic cystic catarrh, is hardly tenable as an explanation of the relation between the two diseases. It may explain the frequency of chronic cystitis whenever prolonged rest in bed is enforced. It is more probable that the increased amount of uric acid in the urine is responsible for the irritation of the mucous lining of the bladder. This, however, does not cause the senile form of chronic cystitis in which there is an absence of glairy mucus found in the ordinary form. The ordinary form of chronic cystitis may occur in the aged either as a sequel to an acute cystitis, or to the irritation produced by a stone or growth, or by the urine the character of which had been altered through retention, increased acidity or by abnormal constituents.

Symptoms.—In senile cystitis there are the usual symptoms of senile degeneration the presence of pockets being revealed by the immediate finding of some more urine in a bladder which had been thoroughly emptied by catheterization; especially is this

the case if the patient changes his position rapidly from side to side and is recatheterized. In addition to these symptoms there is a constant dull ache, increasing upon pressure or whenever the bladder is distended. This ache comes on gradually, is never severe but persists after the bladder is emptied. The urine is generally turbid, ammoniacal and contains bacteria. In the ordinary chronic form there are the same symptoms of persistent ache and ammoniacal, rarely acid, urine, but the urine contains a glairy mucus, and sometimes pus, and there is frequently ulceration of the mucous membrane with more pronounced pain and painful pressure points. There is also in both forms a frequent desire to urinate though the bladder contains but a few drops of urine. The ordinary form gives a history of stone, growth, or an acute cystitis. In rare cases there is a history of urethritis, more often one of gout or chronic rheumatism.

Treatment.—In the senile form of cystitis the treatment is primarily that of degeneration or hypertrophy. The bladder should be frequently emptied, care being taken to thoroughly sterilize the catheter. Catheterization can be entrusted to the patient with strict injunction as to cleanliness. A urinary antiseptic, preferably hexamethylenamine, should be given in 5-grain doses three times daily until the disappearance of turbidity shows that the urine is free from bacteria. Oil of sandalwood in 10-minim doses three times a day will relieve the irritability of the bladder. Irrigation is of little service in this form of cystitis except as a momentary cleansing wash. A 2 per cent. solution of boric acid or a 4 per cent. solution of sodium borate can be used, but the stronger silver solutions are contraindicated.

MODIFIED DISEASES OF THE SKIN

While almost all affections of the skin present peculiarities in the aged due to the senile changes of the skin a few present such pronounced modifications that they will be described separately. In this group will also be placed diseases which occur most frequently or exclusively in the aged and which would therefore belong to the fourth group (preferential diseases). Some of these diseases as well as the benign and malignant

growths are apparently perversions of the normal senile changes in the skin. The true senile diseases of the skin are senile pruritus, the vascular group, senile purpura, and senile gangrene, the benign growth group, senile angioma, sebaceous nevi, warts, keratoma and cornua, the malignant growth group and the senile changes in the hair, nails and glands.

As it is impossible to harmonize the divergent views of dermatologists as to the etiology or pathology of diseases giving similar clinical symptoms, and as the chaotic state of the nomenclature still further tends to confusion, it was deemed best to adhere to the nomenclature adopted by Jadassohn whose work in senile dermatoses is best known. His views are generally followed where they do not conflict with the views of the author.

(A frequent source of error in dealing with senile affections of the skin is senile pruritus, unassociated with the lesion, but which, occurring in the same region, would make it appear as a pruritic affection. The history of a possible antecedent pruritus should be sought for in every case before deciding upon a diagnosis.)

SENILE PURPURA

Senile purpura appears in two forms. Transitory senile purpura occurs as hemorrhagic macules or papules on the lower limbs of aged individuals in whom there are local circulatory disturbances such as varicose veins or generally weak circulation. The spots appear after prolonged standing or walking and disappear when the limbs are at rest in a horizontal position. It is probably due to capillary engorgement brought about by gravitation and weakened return circulation. The affection is insignificant, producing no distress, and is not amenable to treatment. Yellowish discolorations may remain for some length of time.

Permanent senile purpura occurs most frequently on the back of the forearms and hands, especially in those accustomed to work with the forearms exposed. It begins in brick red spots which increase in size and become confluent. Later they are surrounded by a reddish border while the macules become darker and assume the color of blood. After a few weeks the color becomes brown and turns gradually lighter until after

a few months the spots have disappeared entirely or leave but a slight discoloration. The eruption occurs in groups and may reoccur at irregular intervals. The disease is probably due to ruptured degenerate capillaries with transudation of blood into the surrounding tissue. It produces no distress, does not affect the health and nothing can be done for it. Slight trauma may produce serious hemorrhage in these cases. Purpura facticia senilis, described by Jadassohn, is a purpura produced on the back of the forearm when a rough object is rubbed over it. It resembles the permanent form.

SENILE ANGIOMA

Senile angioma or capillary varix is a frequent affection of the aged.

Etiology.—Its etiology is unknown but it occurs most frequently among those much exposed to variations of temperature without sufficient protection. It is more often met with in the country than in the city.

Pathology.—The angioma consists of a minute mass of dilated capillaries lying under the epidermis and sometimes imbedded in the deeper layers of the derma. It thus forms a cavernous body filled with blood. In rare cases the blood is coagulated forming thrombi.

Symptoms.—The angioma appear as dark red, round or oval macules rarely larger than a pin's head. If much larger they may be felt as smooth elastic papules. If deeply situated the color may be purplish or blue and under glass pressure the color becomes lighter but does not disappear entirely. The favorite location is on the back and chest but they may appear in other localities and there is no regularity in their distribution. A senile angioma on the free border of the lips is described by Pasini. It occurs generally as a solitary lesion on the lower lip near the median line, the color is dark red, and there are sometimes fine branch-like projections. The angioma produce no distress, and the patient is often unconscious of them. They may persist for years and do not affect the health.

Treatment.—Nothing need be done for angioma. Should their removal be desired for cosmetic reasons electrolysis, thermocautery, galvanocautery or other cauterization will

effect a cure. There is, however, danger of producing more serious skin lesions.

SENILE SEBACEOUS NEVI

These are white or yellowish, rather hard papules, the size of the head of a pin or slightly larger generally found on the forehead and occasionally on the cheeks and nose. They are sometimes covered by fine telangiectases. They usually contain two or three enlarged pores, often topped by comedones, from which sebum can be expressed. It is a question whether they are nevi or simply hypertrophied sebaceous glands. They are insignificant lesions, producing no distress nor interfering with health. They are important, however, from a diagnostic point as they may be mistaken for soft nevi, molluscum contagiosum, milium, warts or beginning epithelioma. The diagnosis is based upon the expression of sebum and the ability to compress the papule afterward.

No treatment is required except on cosmetic grounds. A simple method is to squeeze out the sebum, wash the spots with alcohol, then apply a solution of tannic acid. If this fails, excision or electrolysis must be resorted to.

SENILE KERATOMA

Senile keratoma, called also precancerous senile keratosis, senile dandruff, concrete sebaceous acne, etc., is a warty growth occurring generally upon the face and dorsum of the hands, rarely upon the neck or scalp. Its etiology is unknown. It consists of a hyperplasia of epithelial tissue, the corneal layer is thickened, the prolongations of the rete extending further into the corium.

Symptoms.—Senile keratomas begin either as a small yellowish, reddish, or brownish, dry plaque, or as a slightly elevated papillomatous wart. The plaques gradually and slowly increase in height and sometimes in size, ranging from the head of a pin to a bean or larger; they are granular or rough to the touch and are covered by fine dry or fatty scales. The upper layers of the scales are easily removed but the lower layer is closely adherent and if removed leaves a bleeding surface, granular and but slightly elevated, or even depressed below the level of the skin. In advanced cases the keratoma occasionally appear as masses

of large crusts covered by scales. They may remain unaltered for years; sometimes, however, one may spread, the tissue becomes indurated, ulcerates, bleeds readily, and assumes the character of an epithelioma.

It is often impossible to determine whether the growth is a senile wart or a precancerous keratosis, yet this is of great prognostic importance. It is safe to say that the wart never becomes malignant except after traumatism, prolonged irritation or some such cause as is usually recognized as an exciting factor in the causation of epithelioma. The senile keratoma may become malignant without any known cause. Owing to the extreme rarity of epithelioma following warts, this prognosis can be virtually disregarded, while in keratoma it must be kept in mind. The differential diagnosis is therefore of importance not alone on account of the prognosis but also on account of the treatment, warts requiring virtually none, while keratoma on account of the possibility of its transformation into a malignant growth should be treated as soon as recognized. Diagnostic points are the location, the origin, and the presence or absence of scales.

Treatment.—Keratomas should be removed. This can sometimes be accomplished by inunction with a 50 per cent. resorcin ointment, but its action is slow. More rapid and more effective is radiotherapy, the usual method of treatment to-day. If the growth becomes malignant total extirpation by the knife is necessary.

Cornua Cutanea.—Cutaneous horns are growths which on account of their prominence have been called horns. They may be of the nature of warts or keratoma, but start most frequently from a plaque of the latter. The surface, consisting of the horny layer of epithelium, is hard, the interior is soft, either fatty, or friable fasciculi composed of cells of the rete. The cornua filiforme is a thin warty growth sometimes found on the eyelids. It is from 5 to 10 mm. in length, 1 to 2 mm. in diameter, and is of no importance. The ordinary form is to be treated as a keratoma.

SENILE WARTS

Senile warts are among the most frequent dermal affections of old age, occurring mostly after the fiftieth year and increasing

in number with advancing age. Their etiology is unknown but it is supposed to be due to some perversion in the normal senile degeneration of the rete mucosum. There is a hyperplasia of the rete, the prolongations projecting downward into the corium, the papillæ extending upward into the excrescence produced by the rete and its epithelial covering.

Symptoms.—Senile warts are soft, flat, pigmented and usually of a granular appearance. They generally range in size from a pin's head to a bean, but occasionally they are much larger. They are round or irregular in shape and usually extend but little above the surface of the skin. The apex may be smooth or finely granular, occasionally coarsely granular or rough and the whole growth may be closely adherent to the skin, like a plaque, or attached by a broad neck, rarely by a thin pedicle. The wart can be removed by a sharp spoon or by the finger nail, leaving a granular bleeding surface which is soon repaired. The favorite location for senile warts is the back, but they may occur upon the chest, neck, abdomen, occasionally on the face, scalp, or extremities. Occurring on the face or on the dorsal surface of the hands, they may be senile keratoma instead. (See Senile Keratoma.)

Treatment.—The senile warts sometimes disappear without treatment. If their removal is desired, any of the ordinary mechanical or medicinal caustics can be employed, care being taken not to injure the surrounding skin.

ROSACEA

The rosacea of Jadassohn is the disease usually described as acne rosacea, not the simple rosacea in which there is telangiectasis without seborrhea. Rosacea is a senile disease since it rarely occurs before the forty-fifth year.

Etiology.—The cause is unknown. It occurs most frequently in those who use alcohol to excess and it is often found in those suffering from digestive disorders. It also occurs occasionally in women during or after the climacteric.

Pathology.—Rosacea is a hyperplasia of the sebaceous glands with retention of sebum, and dilatation and anastomosis of the surrounding capillaries. An inflammatory or pustular acne eruption and comedones appear frequently as a complication.

Symptoms.—Rosacea appears most frequently about the nose, occasionally on the cheeks, chin or lips, rarely elsewhere. It begins with a pinkish erythema gradually getting darker and later minute blood-vessels are seen ramifying over the surface. The surface feels greasy and the dilated openings of the sebaceous glands are capped by particles of dust. A minute mass or string of sebum can be expressed. In the second stage of the disease papules or nodules arise from the erythematous surface. These range from a pinhead to a pea in size, are firm, dark, painless and present minute tortuous blood-vessels. In some cases the nodules grow to the size of a walnut, in rare cases they become still larger forming rhinophyma tumors. These may be single or multiple, round, lobulated or pendulous masses, dark red in color, painless, but producing a conspicuous deformity. According to Kaposi the rosacea nose of the wine drinker is bright red, of the beer drinker cyanotic or violet, of the liquor drinker it is dark blue.

Treatment.—The underlying cause must first be removed if possible. Of special importance in internal medication is the cure of gastric and intestinal disorders. The local treatment must meet two indications, the cure of telangiectasis and of the glandular hyperplasia. The former condition can be sometimes relieved by hypodermic injection of a tenth (1/10) per. cent. solution of adrenalin, or by the local inunction of an ointment of the same strength. If this fails, either the negative galvanic current may be used as in the treatment of hypertrichosis, or a fine thermocautery, or the knife slitting the vessels. Operative work about the face of the aged is often followed by worse disfigurement and sometimes by ulcers and cicatrices which may develop malignancy. Gottheil recommends in the early stage of the disease a mild sulphur lotion and internally ichthyol in 1- or 2-grain doses after meals. Zeller recommends that the lesion be painted with the tincture of the chloride of iron twice daily for five days or until a thick crust is formed and considerable inflammation results. Sulphur ointment is then applied until the inflammation disappears when the painting with iron is to be repeated. Zeissel claims to have cured cases by this procedure. An effective means for diminishing the glandular enlargement is by the use of tannic acid. The surface is first thoroughly washed with alcohol, then pencilled over with

a solution of tannic acid. Resorcin is sometimes effective. For the tumor growth radiotherapy has given the most satisfactory results.

DERMATOSES WITH MINOR MODIFICATIONS IN OLD AGE

Eczema

The various chronic eczemas are of frequent occurrence; the acute form appears seldom, and then mostly about the genitals and lower limbs after scratching. The disease is more tenaceous in the aged, the itching is more severe, the scale and crust formations are more profuse. The acute hyperemia, diffuse edema and serous exudation are less pronounced but there is frequently a sero-sanguineous or purulent sanguineous discharge with intense pruritus. In many cases the eczema follows scratch lesions and eczema intertrigo is frequently met with in the aged. The eczema following scratches appears usually as linear hemorrhagic lesions which may leave small ulcers or if situated over veins they may cause phlebitis or thrombosis. After the crusts disappear their site is marked by grayish or brownish lines. The diagnosis of eczema in the aged is simple. The most frequent type, eczema rubrum, presents the typical symptoms, redness, swelling, infiltration, exudation and crusting with pruritus. When occurring as a linear lesion following scratching it might be mistaken for herpes, but the latter disease occurs only as large vesicles following a nerve trunk or the area of its distribution, it is painful, and the pain is persistent remaining sometimes for months after the vesicles disappear. If itching is present we must determine whether it is a senile pruritus or due to this disease. (Zoster senilis is a far more serious disease and will be considered separately.) *Impetigo contagiosa* may simulate eczema but is rare in the aged, it occurs as discrete vesicles and the crusts appear as if stuck upon the skin. The vesicular type of eczema may be mistaken for erysipelas, dermatitis venenata or syphilis, but the history will generally suffice to clear up the diagnosis.

Acne has no itching, the infiltration is usually limited to the sebaceous glands and comedones are found among the pustules.

Dermatitis is generally of traumatic origin, occurs suddenly

and subsides upon removal of the cause. Simple erythema, due to hypermia, has no exudation. Where exudation does occur it is always an eczema. In herpes febrilis the vesicles are grouped about mucous outlets and occur during febrile affections. Lichen rubrum is rare in the aged and it can be readily differentiated from eczema by the dilated orifices of hair follicles when the scales covering the papules are removed. Psoriasis, also rare in the aged, may be mistaken for papular eczema after scale formation takes place, but the defined contour, abundance of shiny scales, absence of moisture and single type of lesion should clear up the diagnosis. The presence of senile pruritus sometimes makes the diagnosis difficult. This must be excluded.

Treatment.—In the treatment of eczema and in almost all dermatoses of old age, we find some difficulties that are not present in earlier life. The skin being dryer and surface circulation poorer, drugs are not absorbed as readily, they are not as active, the healing process is slower and irritants are liable to produce necrosis and gangrene. The epidermis being thinner and dryer is readily rubbed off, leaving an excoriated surface which becomes a good field for pathogenic germ propagation, hence the frequency of purulent and erysipelatous infection. The uncertainty of the underlying cause in most dermatoses necessitates generally a purely empirical method of procedure. In some cases regulation of the diet will effect a cure, in others protection from exposure to dust or water will suffice, in still others internal medication is required; sometimes a dermatosis will disappear without treatment, in other cases nothing apparently helps. In the treatment of eczema the cause should be determined if possible and eliminated. This can usually be done if there is a local cause, as scratching, intertrigo, vocational irritation or a parasite. If no cause can be discovered attention must be paid to the ordinary dietetic and hygienic rules and we must depend upon the local medication. Water should be excluded except in the inflammatory stage when clothes dampened with a weak solution of lead or aluminum acetate can be used. After the inflammatory stage has passed an alcoholic solution of 1/4 per cent. of thymol should be applied for an hour several times daily while in the intervals, the surface should be covered with zinc stearate.

Other drugs that are of service in some cases are zinc, lead and bismuth salts, white precipitate, ichthyol, tar, pyrogallol, salicylic acid, sulphur, etc., in powder or ointment. Radiotherapy, massage, and other mechanical measures have been tried with occasional succes.

Pityriasis tabescentium, in which fine scales of dried epithelium can be rubbed or scratched off the skin, is a normal senile condition, not a disease. Other forms of pityriasis are extremely rare in the aged. *Prurigo senilis* is described by Pic as "La maladie cutanée la plus commune chez de Viellard est le prurigo." On the other hand Jadassohn declares that the true prurigo, *prurigo Hebrae*, does not occur in the aged and is furthermore a rare disease at any period of life.

Psoriasis when occurring in the aged is usually carried over from earlier life and does not differ essentially from the earlier disease. The scaly formation may be less profuse and the lesions may be lighter in color and occasionally there is itching, probably a local senile pruritus. In the treatment, which is purely empirical, irritating drugs must be avoided.

Dermatoses due to mechanical, chemical or thermic causes occur in the aged less frequently than in earlier life, the aged being less exposed to them. *Corns*, *bunions*, *hammer toes* and other pedal defects are frequently found and present no difficulty in their diagnosis. Bedsores occur frequently and may become gangrenous. The diagnosis is simple.

Burns are far more serious in the aged on account of their constitutional effect and the slow local regeneration. Attention must be paid to the heart and kidneys apart from the local indication.

Pernio is rare. Dubreuil described a form of *chronic senile pernio* occurring in the aged, not accompanied by bullæ or ulcers but by circulatory disturbances which become pronounced with the advent of cold weather and gradually disappear in the spring. The parts affected become tumefied, irregularly marked with dark red or violet marbling, and are numb. During the height of the symptoms the parts are intensely painful, swollen and dark. This condition is, however, rare.

Vocational dermatoses are infrequent and present no marked difference from such conditions in maturity. *Toxic dermatoses* probably constitute the majority of senile dermic affections

but the etiology of many is still disputed. It appears certain that the same toxic cause may produce various lesions and diseases and the same disease may be due to a variety of causes, toxic or non-toxic. It is consequently impossible to make a definite classification of toxic dermatoses except where the etiology is positively known, such as urticaria and erythema multiforme, dermatitis herpetiformes, pemphigus, etc. *Parasitic dermatoses* are infrequent in the aged, perhaps because they are less exposed, or less hairy, or because the senile skin is a poor field for their propogation. When they do appear they do not differ from the diseases of maturity. Pediculi vestimentorum occur occasionally among the same class as in maturity, but do not present any difference. Parasitic disease may be mistaken for senile pruritus, and the scratching may result in eczema. *Mycoses* are rare in the aged and do not differ from the mycoses of maturity. A body favus is occasionally seen shortly before death where there had been a prolonged cachexia, as carcinoma, tuberculosis, etc. Jadassohn believes it is due to an abnormally located mycelium growth.

Bacterial dermatoses occur rather frequently. (The most important of these, erysipelas, will be treated among the infectious diseases of the fifth group.) The pyodermatoses are seen occasionally either as primary affections or secondary to other diseases, the latter sometimes predominating over the original disease. The streptogenic dermatoses appear in two forms, impetigo contagiosa and ecthyma. The true *impetigo contagiosa* does not occur in the aged but a mild vesicular eruption of streptogenic origin is occasionally found as a complication of other dermatoses. These present superficial vesicles which rupture, forming transparent yellowish crusts, and produce no constitutional effect. *Ecthyma* occurs occasionally as a secondary disease in pruritic affections. It is probable that the mild impetigo is the outcome of a non-virulent strain of streptococci, while ecthyma is the result of a virulent strain of these germs. The latter may begin as a vesicular eruption, soon becoming pustular, or it may begin with pustules which, after rupture and drying, produce opaque yellow scabs. They are more deep seated than the other and may cause ulcers. Local antiseptics are indicated. The *staphylogenic dermatoses* generally attack the glandular orifices and tubes, producing inflammation and

abscess. The sudoriparous glands are apparently immune from attack in the aged, but the sebaceous glands and hair follicles are occasionally the seat of inflammation from this source. More serious affections due to staphylococcic infection are boils and carbuncles. The simple *folliculitis superficialis* consists of numerous small pustules upon an inflamed base. It follows local irritation with infection, gives little distress and may persist for weeks or months if not treated.

Furuncles may follow a folliculitis as an extension of the pyogenic process, or it may begin as a more intense and extensive folliculitis or a perifolliculitis. In this as in all pyogenic affections of the skin there must be a peculiar state of the system to furnish a suitable field for the existence and development of the germs. This is found in diabetes, nephritis, gastric or intestinal disorders causing autointoxication, circulatory diseases, etc. Diabetes especially favors the production of furuncles and carbuncles.

Carbuncles are virtually deep-seated furuncles of large size in which the integument is undermined and several sacs are formed. These sacs hold pus which makes its way to the surface through one or several openings. There are usually grave constitutional symptoms present. In simple folliculitis antiseptic lotions or ointments and the observance of dietetic and hygienic rules generally effect a cure. If crusts have formed these must be removed. In furunculosis the treatment of the cause is of primary importance. If it is a chronic disease like diabetes or nephritis there may be successive crops which must be dealt with. Occasionally internal medication with arsenic is of service. Serum (vaccine) therapy has given brilliant results when used at the onset of the disease but after the boil has developed it is useless, as the slough must be removed before healing can be effected. A furunculosis vaccine is on the market with reports of remarkable results in furuncles and boils. Furuncles can sometimes be aborted if the cause can be rapidly eliminated. If due to autointoxication following constipation, active catharsis is sometimes effective and this is the rationale of magnesium sulphate treatment, which is occasionally curative. If this fails, excision or the cautery becomes necessary. Carbuncles must be treated like boils, but if the vaccine therapy fails, other abortive measures are futile. The

cautery or the knife should be used early to allow free discharge of the pus and the slough should be picked out with a forceps. The injection of carbolic acid is intensely painful and may produce extensive necrosis. The constitutional symptoms of septic infection as well as the causative condition must not be neglected.

Tubercular dermatoses occur seldom in the aged and then generally as some form of lupus. The *senile lupus* does not differ from the lupus of earlier life. Beginning as a well-defined slightly elevated patch, light brown in color, and about the size of a hemp seed, or slightly larger, it increases very slowly in size, while the center becomes depressed. The top is covered with glistening epithelial scales. It may remain small throughout life, or it may increase to the size of several inches in diameter, it may remain indolent for years or it may expand more rapidly, invading the deeper tissues and becoming nodular and ulcerous at the top, or it may develop into an epithelioma. In cases originating in old age the lesion is often formed rapidly while the further progress is slow.

Earlier lupus erythematodes may suddenly become active and progress to suppuration or to tissue destruction. In some cases favorable involution and scar formation ensues. The favorite location of lupus is the face; occasionally it appears upon the scalp, rarely upon the hands, feet, or other portions of the body. The forms met with in the aged are the lupus erythematodes, lupus vulgaris, and *lupus vulgaris erythematoides* (Lenoir), an intermediate form. The scrofuloderma are rare in the aged and are almost always associated with glandular or bone tuberculosis. Jadassohn describes a *tuberculosis fungosa serpiginosa* which appears to partake of the character of both lupus and the scrofuloderm. This occurs most frequently in the lower extremities in connection with bone or gland tuberculosis. In many cases there are fistulous openings from the underlying lesions and near their openings serpiginous plaques form. These are soft, dark red, irregular patches, which grow rapidly and spread, while their centers form scars. Yellow miliary macules as a result of colloid degeneration are sometimes found in the scar tissue. The tuberculides may be mistaken for syphilides or malignant growths. Where the diagnosis rests between tuberculosis and syphilis if the history is insufficient, it may be neces-

sary to resort to the tuberculin and Wasserman tests in order to obtain the etiological factor. The history, and the slow progress of the tuberculides will distinguish them from malignant growths. The treatment includes the constitutional treatment for tuberculosis and local treatment of the lesion. The Finsten light, radiotherapy and the cautery are the most effective methods of treating the local condition. The older methods by inunctions are seldom used, since the mechanical measures are so much more effective. Where a tuberculous affection is due to an underlying lesion, such as a bone or gland tuberculosis, the latter must be cured before the dermal lesion can be improved.

Lepra may occur in the aged but it does not differ from the same disease in earlier life.

Angioneuroses are sometimes observed in the aged but they do not differ from the diseases of earlier life excepting one form of herpes called *zoster senilis*. In this the symptoms are much more severe and prolonged than in the ordinary herpes zoster, the disease is more deeply seated, the vesicles are increased in number and size and they frequently become transformed into pustular or hemorrhagic lesions. In some pustular cases the pustules ulcerate, in others they form crusts which leave persistent cicatrices. The hemorrhagic form disappears leaving a deep pigmentation or a scar. The pain is intense and may persist for months after the lesion has disappeared. Secondary cutaneous diseases sometimes follow.

In the treatment of zoster senilis the relief of pain is the first indication. Cocaine in 2 per cent. solution or ointment is the most effective local measure, but the relief is only temporary. Orthoform, ointments, antipyrin injections, heat, cold, have all been tried and occasionally give relief; frequently they have no effect. The internal treatment is equally unsatisfactory. The usual antineuralgic measures help in some cases and aggravate the condition in others. In many cases morphine becomes necessary. In this as in other neuroses of uncertain origin drugs and other therapeutic measures must be used empirically, employing each long enough to observe an effect, either physiological or therapeutic. If beneficial we will naturally follow up that treatment, but if detrimental we must go to the opposite class of drugs or other measures.

It is sometimes possible to determine the etiological factor; in such cases the elimination of that factor is of first importance. Other forms of herpes as well as urticaria and simple erythema do not differ from the same diseases in earlier life. They occur in old age under the same conditions and require the same treatment. The *progressive nutritional disturbances* are infrequent. Most of the hyperkeratoses have a congenital basis and, while diseases of earlier life, they may persist until old age. They generally disappear as a result of the senile changes of the skin. Hyperpigmentation and hypertrichosis occur as a primary condition but they present no difficulty in their diagnosis and are of no importance except possibly from a cosmetic standpoint. Scleroderma is rarely seen in the aged.

The retrogressive pathological changes in the skin are mostly variations of the normal senile atrophy. Some, like *diffuse idiopathic atrophy*, *acrodermatitis atrophicans*, *xeroderma*, etc., are extremely rare; others like *cutis laxa*, *striæ*, *vitiligo*, etc., are unimportant and require no treatment, while alopecia and canites have been discussed.

The *epidermolyses*, the *pemphigus* group, *dermatitis herpetiformis*, and *epidermolysis bullosa heredita* are infrequent at any time of life and rare in old age. When they do occur they do not differ from those of earlier life.

Dermal glandular disturbances occur frequently. The sudoriparous glands are subject to hyperidrosis, anidrosis and bromidrosis (which have been discussed), miliaria and hidrocystoma. The latter are retention cysts, bluish and about the size of a pin head, forming in the epidermal opening of the sweat glands. They occur most frequently on the face of elderly women who work in hot rooms. When they perspire the perspiration fills these minute cysts which then become elevated above the surface of the skin. Upon rest in a cool place they disappear. Miliaria appears in the aged as a simple vesicular affection of little importance. It is identical with the prickly heat of children. Diseases of the sebaceous glands with the exception of rosacea are comparatively infrequent. Seborrhea oleosa and sicca occur almost exclusively in the form of rosacea. Comedones and acne are rare, although an acne eruption does occasionally occur in women about the time of the climacteric. An artificial acne may be produced by the staphylococcic infec-

tion of a folliculitis induced by irritating substances such as depilatories, face washes, tar ointment, etc. It disappears if the cause is removed. The *acne sclerotisans nuchæ*, a folliculitis sometimes becoming pustular, is occasionally seen about the neck as a chronic condition carried over from maturity The pustules may be opened but there is no certain method of curing this condition. It sometimes gradually disappears. The *sycosis non-parasitoria* is probably the same condition occurring in the beard. Other forms of acne occur occasionally, but they do not differ materially from the diseases of maturity.

CHRONIC ULCER

Chronic ulcers, especially upon the legs, are frequently met with in the aged. They are generally due to some slight traumatism, a blow, bruise, or scratch, which on account of the poor surface circulation does not heal readily. A chronic ulcer may also be due to a ruptured varicose vein. An ulcer occurring upon a previously healthy skin or upon a keratoma or other growth is generally malignant in spite of its chronic course. It is important before instituting treatment to determine whether the lesion is a simple ulcer, a malignant ulcer or a syphilitic or tubercular lesion. These all may look alike, run a chronic course, gradually enlarging and producing no distress until a sensory nerve is involved. The simple chronic ulcer begins within a few days after the initial lesion, whether bruise, scratch or rupture of a vein. The traumatic lesion does not heal, a crust may form while the ulcer below it persists. If there is a bruise, an abscess may form which will open and leave an ulcerated base. There is usually a serous exudation, or if infected the exudation becomes seropurulent or purulent, rarely serosanguineous. The ulcer grows in extent, becomes slightly deeper, and has sharply defined but not indurated or everted edges. It is painless unless a sensory nerve is involved but there is often itching around the margin. The syphilitic ulcer can generally be eliminated by the history of infection, the primary lesion and the presence of other lesions. The tubercular form may give a tubercular history, it originates in a tubercle which breaks down, its advent is slow and when ulcer-

ating there is little or no pus, thus differing from the ulcer following a bruise. The malignant ulcer has usually a history of a preceding growth or of scar tissue. The simple chronic ulcer does not produce constitutional symptoms unless it becomes infected, and giving no local distress it is often neglected, perhaps for years. If infected, the constitutional symptoms may become grave, while the excessive amount of pus may cause exhaustion. In the treatment of these cases care must be taken to avoid giving the patients pain. We must also remember that granulations will not start in disorganized tissue, however clean we may get it. The surface must be both healthy and clean. The ulcer is first washed with warm water, then a solution of peroxide of hydrogen is applied until bubbling ceases, then warm water must be used again to wash away the H_2O_2. After we have a clean surface a 5 per cent. solution of cocaine is applied followed a few minutes later by a caustic, either chromic acid or carbolic acid or nitrate of silver and washed clean again. The ulcer is then filled or covered with lanoline. The following day the washing must be repeated and after applying the cocaine the slough is removed by forceps or by the knife. An active hyperemia is necessary to initiate healthy granulation and a mild hyperemia is required to keep it up. Without a sufficient supply of circulating blood these chronic ulcers will not heal. Small dry cups or the application of hot dry or moist cloths will generally bring enough blood to the surface to produce the required hyperemia, for the starting of granulations. But no granulations will form if the surface is covered with any substance which disorganizes tissue, therefore if caustics or even but a mild bichloride solution has been used the surface must be washed clean and if necessary abraded. A nuclein should be used as a dusting powder to stimulate granulations and the ulcer should be packed with an animal fat preferably anhydrous lanoline. If this method is followed the ulcer can be cured, providing there has been no infection. Should pus continue to flow in spite of such repeated washing showing a more extensive or a general infection, serum treatment may be necessary before local treatment will be effective.

(*Note.*—Nuclein containing sugar of milk is intensely irritating.)

NEOPLASMS

Benign Growths

Of the benign growths, *warts*, *nevi* and *fibromata* are the most frequent ones in old age. (Senile warts and senile sebaceous nevi, have been described.) It is probable that the senile sebaceous nevi are really adenomata. *Nevi* are almost without exception carried over from earlier life, persist unaltered and aside from their unsightliness produce no distress. Nothing need be done for them. *Fibroma* occur frequently and may appear in numbers upon the neck and upper part of the chest, less often upon the face or extremities. When small they may lie entirely beneath the skin or project slightly as circumscribed nodules or plaques; when larger they appear as buttons or become pendulous. They vary in size from a pea to a mass weighing several pounds, are covered with normal skin, produce no distress and if left alone they will remain unaltered after they have reached the limit of their growth. Small pedunculated growths can be clipped off and the pedicle cauterized. Sessile growths should be left alone unless their size or location makes removal advisable. The surgical procedure for their removal depends upon the preference of the surgeon. Electrolysis, radiotherapy, galvanocautery, thermocautery or the knife can be employed. *Lipoma* like fibroma is seen in the aged, often as small pendulous tumors. They resemble fibroma but are softer, more regular in shape and generally appear singly or in groups of two or three, rarely in numbers. *Keloid* and *xanthoma* are rare and when occurring are generally carried over from earlier life. Keloid may, however, follow a traumatism. Other benign growths like hard warts, mollusca contagiosa, condylomata, etc., rarely or never occur in advanced age. In dealing with benign growths it must be borne in mind that in some cases they become malignant and that their transformation into a malignant growth sometimes takes place after operation. Better results are apparently obtained by the X-ray and Finsten light than by the knife. Radium therapy is still too uncertain and too limited in its distribution to be more than an experimental measure and the same applies to carbonic acid snow. Thiosinamin, fibrolysin and scarlet red have given good results in some cases and fail completely in others.

MALIGNANT NEOPLASMS

The most important of the malignant growths in the aged is the epithelioma. It would serve no purpose to discuss the numerous theories that have been advanced to explain the etiology and pathogenesis of cancer. There are also diverse classifications based upon structure, clinical manifestations, tissue involved, primary or secondary appearance, etc. The primary dermal epithelioma originates as a dermal lesion; the secondary growth is due to an underlying cancer as of the breast, or is an extension from a cancer in some neighboring tissue as from a cancer of the vagina, or it is a metastatic lesion carried by way of the lymphatics or blood-vessels.

The primary epithelioma is, in most cases, secondary to another affection of the skin. Bond says "the complex cell change that we associate with cancer has been built up by variational changes from the normal type and that one of the stages passed through is represented by the various forms of benign growth."

Epithelioma appears clinically in two types, a superficial, mildly malignant, extremely chronic type, and the other deep, active, and rapid. There is no sharp dividing line between the two, and the disease may begin as a superficial chronic lesion which after existing for years suddenly becomes actively malignant.

The active form may find its seat primarily upon apparently healthy tissue or recent trauma or upon the site of a lupus, syphilide, leucoplakia, crural ulcer or scar, rarely upon a senile keratoma or xeroderma. It usually begins as a hard light colored nodule gradually becoming dark red, irregular in shape and but slightly above the level of the skin. There is generally a hyperkeratosis and sometimes a papillomatous growth of the nodule. A few weeks or months later the surface becomes eroded and beneath it there is a raw granular or papillomatous surface which in some cases becomes ulcerative, in other cases there is a more or less rapid growth of the papillomatous tissue which soon extends beyond the surface and forms the classical "cauliflower growth" of malignant papilloma. In this form of epithelioma the morbid vegetation may reach the size of a hen's egg. It is usually spongy, warty and exudes a foul-

smelling clear or sanguineous fluid. After a time fissures spread through the mass, it becomes ulcerative and the whole mass turns into a foul ulcer, penetrating the tissues and expanding on all sides. In cases where the tissues break down and become ulcerated from the start, the further progress is the same as in the papilloma. The ulcer presents a hard, overhanging border which is undermined and from which a semiliquid mass containing epithelial cells can be expressed. The epithelioma can extend through the tissue perforating and destroying muscle, fascia and even bone. In some cases the ulcer exudate forms a crust which becomes hard and completely hides the destructive process underneath. This form of epithelioma becomes painful from the moment that the skin is eroded and the pain becomes intense if the surface is irritated. It bleeds readily and the surface is necrotic. As the disease progresses the neighboring lymphatic glands become involved, later there are metastatic cancerous ulcers, cachexia appears and the constitutional symptoms follow.

The mild superficial epithelioma usually finds its seat upon a senile keratoma. This may exist for years before it is noticed that there is any change in its size or character. In some cases there is nothing more than a small superficial nodule perhaps covered with scales, or a hard crust covering an excoriation or an ulcer produced perhaps by scratching or by a slight blow. This may persist for years without change, or other small nodules may form about the site of the original lesion. Sooner or later the nodules break down and become ulcerated, the ulcers being at first shallow and small but gradually extending and in some cases rapidly destroying the underlying and surrounding tissue. The rodent ulcer thus formed is at first painless, later it becomes intensely painful. Occasionally the destructive process halts and after remaining quiescent for years starts afresh or the ulcer heals. This form of epithelioma rarely involves the glands and produces neither cachexia nor other constitutional symptoms.

The favored location of the deep epithelioma is the face, mouth, lips, genitals and anus, while the mild epithelioma is generally found in the upper part of the face about the eyes or nose.

Paget's disease of the nipples and most epitheliomata found

in other parts of the body are secondary to underlying or contiguous carcinomata. A class of malignant growths which begin in soft nevi are sometimes classed as epitheliomata, sometimes as sarcomata. When arising from pigmented nevi the growth is pigmented producing the melanotic carcinoma. It follows the course of the deep epithelioma, being rapid in its onset and development and speedily involving the lymphatic glands.

The *lentigo maligna* of Hutchinson begins as a darkly pigmented macule which after years of quiescence suddenly begins to give evidence of active malignancy and within a short time acts as an active epithelioma.

The diagnosis of epithelioma is often difficult, as there are several dermatoses presenting similar clinical manifestations without the histological characteristics, while the histological characteristics of the former have been found in benign growths. The diagnosis depends upon the occurrence of both the clinical and the histological findings. A positive tuberculin or Wasserman test does not exclude a coexisting carcinoma. The only tuberculide giving similar symptoms is lupus. This occurs earlier in life, is generally composed of a group of lesions and the border of the ulcer is never indurated or everted. The syphilides present a multiplicity of lesions, they do not spread, are not painful and improve under the usual treatment for the disease. The benign tumors must be diagnosed by the histological findings. Sarcoma is more rapid in its development, occurs earlier in life, rarely ulcerates and involves neighboring tissue or produces metastatic lesions of the same variety. As a last resort, if the diagnosis is still doubtful, the microscope must decide.

Sarcoma

The sarcomatous growths are relatively rare in the aged. The sarcoma is a perversion of connective-tissue cell growth occurring under circumstances very much like an epithelioma. The growth proceeds, however, much more rapidly. It begins as a small nodule several of which appear in the same locality. By increase and confluence they form tumors, sometimes as large as a hen's egg, and may appear on any part of the body.

They are often pigmented and painful, sometimes vegetations appear upon them, occasionally they ulcerate. They may be secondary to sarcoma in another part of the body, or primary, beginning upon the site of a traumatism or other skin lesion, rarely upon a healthy surface. They present various forms, may be hard or soft and show under the microscope characteristic round, spindle-shaped or giant cells. While not exhibiting the local destructive tendencies of active epitheliomata, metastases are more frequent, extirpation is followed by recurrence with increased virulence and the constitutional effects are pronounced.

Atypical forms of epitheliomata and sarcomata are occasionally seen in the aged, but a particle clipped from the growth and examined under the microscope will generally determine its character.

Treatment of Malignant Growths.—There is probably no pathological condition in which more therapeutic experiments have been made than in malignant growths. About everything known to have a caustic or other destructive action upon animal tissue has been used to destroy malignant growths, while internal medication has kept pace with external measures. We have found, however, no better method of dealing with such growths than total extirpation by the knife. Various measures have been employed to bring about the destruction of the growth, yet none of them has been generally accepted. Some still adhere to chemical caustics, others prefer to use the knife. Among the newer measures are radium emanations, the X-ray, high-frequency Herzian waves, Finsten light fulguration and Forest's cold cautery. Each has its supporters, each has accomplished a more or less complete destruction of the growth, yet none has absolutely prevented the metastases, the involvement of any neighboring glands or the recurrence of the growth. At the present moment the tide is turning toward internal medication, the latest method of treatment being chemotherapy. It is sought to obtain "a chemical substance which, administered by the mouth, shall exhibit affinity for the peculiar chemical constitution of the cancer cell. Granting that this affinity produces a result injurious or destructive to the growth, there at once ensues a cure of cancer" (Morton). So far this has not been accomplished. In the treatment of epithelioma

Judd reports about 90 per cent. of cures by the X-ray but in the other 10 per cent. especially in old persons, the X-ray, "while it caused destruction of the growth, failed to prevent its almost immediate recurrence." Korbl reports that of seventy-three cases that were re-examined after X-ray treatment, thirty-seven had a recurrence of the growth. Moullin, Finzi and Dominici reporting upon the result of radium treatment gave few favorable results. The object is always to destroy the growth and as long as the growth is a purely local condition without gland or constitutional involvement, the simple caustics like chloride of zinc, caustic potash or soda, acid nitrate of mercury, lactic acid, etc., will suffice. Arsenic is still the most popular of this class of drugs, although its action is apparently not that of an escharotic but of a toxin to the pathological cells. The great danger in local arsenic medication is arsenical poisoning through absorption. This can hardly be prevented, a weak solution or paste being useless. We must, therefore, use it in a strength of at least 1 per cent. to be effective. This, if used for a long period, produces toxic symptoms and the treatment must be discontinued or replaced by the use of escharotics. For deep growths the only reliable treatment is early and complete excision. Some surgeons go further and excise neighboring lymphatic glands. Even this generally fails, if the growth is a sarcoma, as metastatic growths almost invariably follow. At present we have no means of combating this form of growth and all that can be done is to destroy the growths as they appear, relieve symptoms and maintain the strength of the individual. It is only in the superficial forms of epithelioma that we may be reasonably certain of effecting a permanent cure. In the deeper lesions, especially after glandular involvement, operative intervention may give temporary relief, but it rarely prevents a return of the disease. The one imperative rule in all cases is to remove the growth completely as soon as its character is established.

SENILE PSYCHOSES

Psychic disorders occur frequently in the aged. **Senile dementia** is by far the most prevalent, being in many cases secondary to other disorders, and generally the terminal stage of all

mental diseases that are carried over from earlier life. The primary apathetic senile dementia occurring as the end result of the normal senile degeneration of the brain and of cerebral softening has been described under those heads. Secondary forms are described by some authorities as agitative senile dementia occurring during or following mania, paranoiac, melancholic, hypochondriac, etc., senile dementia. Other forms of senile dementia are due to traumatism, apoplexy, or arteriosclerosis, and the terminal dementias of other psychoses. In the secondary dementias the primary psychosis gradually becomes milder in its manifestations while the intellect becomes duller and duller until mentality is completely obliterated. Senile dementia whether primary, secondary or terminal is progressive and incurable.

Acute senile dementia described by Salgo consists of a rapid dementia following acute manifestations of mental impairment, dulling of the intellect, incoherence and confusion, loss of memory, etc., with constant restlessness. These are accompanied by visceral disorders, insomnia, and rise in temperature. It may clear up or the dementia may become progressively deepening.

Amentia, senile delirium or hallucinatory confusion is occasionally met with. This generally begins with rapid confusion, loss of orientation and great restlessness, followed by illusions, delusions, hallucinations and phobias, the patient is excited and violently active, with periods of comparative quiet during which the mental phenomena are milder and the restlessness disappears. There may be delusions of persecution or of grandeur, violent outbursts or depression. In many cases the reflexes are exaggerated, pupils irregular, there is a weak irregular, rapid pulse, fever, constipation, and icterus. Albumin and indican are found in the urine. Amentia may terminate in dementia, occasionally it is the precursor to a hemiplegia. Recovery is rare in the aged. While the violent symptoms may abate there is a progressive dulling of the intellect until dementia is complete. A form of senile delirium sometimes occurs during the senile climacteric.

Hypochondria and **melancholia** are so frequently associated and so intimately connected in the aged that they will be considered together. The melancholia may follow a neuras-

thenia, psychasthenia or hypochondria or it may be a primary condition due to some powerful emotion. Hypochondria also may be primary or secondary to a neurasthenia or psychasthenia. There may be emotional depression without impairment of the intellect or such mental impairment as is usual with the normal senile degeneration, but there are always unnatural fears, or a haunting anxiety without a definite object. In some cases there is a fear of disease, of death or future punishment for insignificant misdoings. The hypochondriac is given to introspection and to self-examination of his physical condition. The discovery of an abnormal feature, a macule or papule, a slight rise in the rate of respiration or pulse rate will suffice to arouse the most agonizing fear of disease, culminating in melancholia. Slight symptoms are exaggerated and suggestion or mimicry will give rise to imaginary symptoms and sensations. In some cases it will be possible to explain away symptoms, but in most cases the efforts of the physician to quiet the patient's fears are looked upon with suspicion. When melancholia supervenes it is impossible to make the patient realize the absurdity of his ideas and fears, but it is often possible in an early stage of melancholia to make him forget them. He will greet the physician with numerous complaints and the latter, if tactful, will turn the patient's thoughts to other subjects. He will forget then his ailments and may, when reminded of them, forget their location.

Senile melancholia may appear in an apathetic, depressive form or in a restless, agitated one. In the apathetic form the patient will sit for hours, apparently indifferent to his surroundings, complaining, mumbling or weeping. In the agitated form the patient is restless, excited, anxious, fearful, and sometimes violent. In the violent state he may commit murder or suicide. In some cases there are remissions during which the patient is comparatively free from the mental and emotional depression but each attack leaves the mental faculties more impaired and finally the patient sinks into a dementia which is progressively deepening.

Treatment.—In the treatment of senile psychoses we must bear in mind the presence of senile degeneration of the brain with the certainty of present or ultimate senile dementia. The keynote of treatment is psychic stimulation. This is opposed

to the generally accepted method of treating psychoses by sedatives, rest and quiet. When there is much restlessness, warm baths should be employed and if these fail we may resort to the bromides. Mental confusion is best treated by powerful but harmonious sensuous impressions which will attract and hold the patient's attention. An old familiar air will sometimes dispel the confusion and this is one of the most effective means for stimulating memory and quieting an excited patient. It is often possible to reason with a patient while his mind is so diverted. In some cases the conversion of the subject of an hallucination into the reality, unknown to the patient, will restore reason. A patient who nightly saw a ghost at the foot of his bed was cured of this hallucination when one of his friends suddenly appeared at the foot of the bed, covered with a sheet, then threw off the sheet and spoke to the patient. Sometimes a powerful impression will destroy an illusion, delusion, hallucination or phobia. The following is a typical example. The patient aged sixty-nine who had been an ardent fisherman in his earlier years, was suffering from arthrosclerosis of the ankles and shoulders. He had been treated for chronic rheumatism, but the condition grew worse and he feared that his joints would all grow stiff and he would become like the ossified man he had seen in a museum. From this fear he developed the dread that he would become a burden to his family and that they were anxious to get rid of him. He then developed the apathetic form of melancholia. Some of his friends took him then upon a fishing boat and a line was placed in his hands. At first he was indifferent to his surroundings until there was a sudden tug upon his line. He was startled for a moment but as soon as the line was passing through his hand he grasped it and pulled in his fish. He continued fishing and returned home in a cheerful spirit and cured of the melancholia.

Notwithstanding the benefit of change of environment and the constant attention of physicians and nurses, incarceration in an asylum is perhaps the worst possible treatment for senile psychoses. The association of the senile dement with other insane persons will not improve him mentally but may produce mental perversion in addition to mental weakness. Such patients need constant diversion and mental stimulation, not rest and quiet. Mental agitation requires stimulation of a different sort

and the more intense the excitement the more intense the stimulation must be. A brass band playing a loud patriotic air will attract attention where a violin solo will have no effect; the harmonious movements of a large ballet will quiet the mind while the confused movements of dancers in a ball room will disconcert and irritate the patient. A large well-drilled chorus presenting pleasing stage pictures will relieve melancholy and depression and will calm mental agitation by substituting another form of mental stimulation. Aural stimulation, especially by old familiar airs, is more effective than visual stimulation unless the latter can be prolonged and the interest maintained. The stimulation may be prolonged until brain fag sets in when the patient will fall asleep. Medication must have the same purpose as the psychic measures, mental stimulation. The only drug suitable in these cases is phosphorus given in 1/50-grain doses three times a day.

MODIFIED PSYCHOSES

General paresis is rare in the aged. When it does occur it does not give the clearly defined clinical picture that it presents in maturity and it may be mistaken for senile dementia. In general paresis in the aged the delusions of grandeur are less florid than in maturity, but they appear early and thereby distinguish this disease from senile dementia. There may be delusions of grandeur in the delirious form of senile dementia, but these are generally combined with unsystematized delusions of persecution, phobias, and weakened intellect. General paresis in the aged develops more rapidly but the apoplectiform attacks occur less frequently than in maturity. There is the same difficulty in the speech, which is rapid and irregular, the patient sometimes hesitating, at other times tripping, running one word into the next without a break between them, or suppressing words or syllables. The paresis and paralysis of the extremities occur at irregular intervals and clear up partially, but each apoplectiform seizure leaves the patient mentally and physically weaker than before. There is no known treatment for this condition.

Mania is rare except in the maniacal outbursts of delirious senile dementia, in the form of a few monomanias peculiar to

the aged, as oikeiomania, and in circular insanity. There are generally delusions of grandeur and morbid impulses arsing therefrom, the latter being often immoral or criminal. There is a disregard of consequences to others and of retribution or punishment to himself. Mental agitation is pronounced. Mania in the aged sometimes disappears for months, sometimes reappearing without apparent cause and in a more aggravated form. Some cases are succeeded by amentia or dementia. Mania alternating with melancholia and lucid intervals characterize **circular insanity.** Such cases are occasionally met with in melancholia when some insignificant mental or physical irritation will cause a maniacal outburst sometimes lasting for days, followed by exhaustion. During this period the mind is apparently clear but mental depression follows and the cycle is resumed. In rare cases the melancholia follows mania, the latter following a clear period. There is frequently a history of early mental disorder, hysteria, neurasthenia or other neurosis. **Senile paranoia** with delusions of persecution occurs frequently, yet it is seldom recognized. In most cases the sense of hearing is lessened and the patient, realizing his diminished usefulness, becomes suspicious when conversation which he cannot hear is conducted in his presence. This gives rise to delusions of persecution, the patient fearing that those who have the charge of looking after him are anxious to put him out of the way. Delusions of smell and taste arise from the fear of being poisoned and auditory and visual illusions develop from other fears. The patient exaggerates his own importance until his ideas about himself assume the shape of delusions of grandeur and while boasting of his strength and ability to stand pain, he will complain of the intense pain associated with insignificant hurts. In some cases this is due to the desire to arouse sympathy, generally, however, to hypochondria.

When the fear of persecution is directed to a member of the family there is generally a substantial basis which in itself is trivial, such as momentary absence, food too hot or too cold or too salty, a sharp reply or reproof, etc. The patient broods over this, exaggerates its importance, and develops suspicion and hatred, fear is aroused and this is converted into delusions of persecution. The fear that his enemy may kill him if he utters any complaints will prevent the patient from expressing

his fears and the first intimation of the patient's mental condition may come when his will is read. The tactful physician will often be able to obtain the confidence of his patient sufficiently to elicit paranoiac delusions, although he will not be able to remove them. A querulent form of paranoia is met with occasionally after the senile climacteric and as mental decadence proceeds the complaining and whining gradually give way to a *mumbling* dementia.

SENILE PSYCHASTHENIA

Psychasthenia or mental exhaustion is generally associated with neurasthenia. Owing to the failure to differentiate between the two conditions, psychasthenia is usually considered as the cerebral phase of neurasthenia. Psychasthenia may, however, exist without nervous exhaustion or weakness but will give symptoms of the latter. When the impulses originating in the brain are weakened they are carried more slowly and with less force, by the nerves and thus the nervous symptoms are produced. Occurring in the aged it presents slight differences from the similar condition of maturity.

Etiology.—Psychasthenia is due to excessive mental activity with insufficient repair and to the probable absorption of the waste products of mental activity. It occurs most frequently in those engaged in exciting mental work, especially where rapid action or mental concentration is involved, hence we find it frequently in professional men, writers, ministers, physicians, and scientists, in those who must calculate and reason quickly as brokers and others engaged in buying and selling without long deliberation, in bookkeepers, etc. It occurs generally when there has been a period of intense mental activity following a period of rest. After mental deterioration has begun in the aged, even slight excitement will suffice to produce brain fag and if this excitement continues brain exhaustion may result.

Symptoms.—The symptoms can be divided into three stages, a preliminary stage, a stage of brain fag and a stage of brain exhaustion.

During the preliminary stage the mind is in a state of tension. Ideas whirl or fly through the brain and the individual

cannot express himself fast enough. If he writes he omits the last letter of the word or he omits words altogether. He makes errors in calculation by overlooking figures, errors in speech by chopping off words and phrases. He does not take time to deliberate where deliberation is necessary, forms extravagant projects, losing the sense of time and space. In this period he is in a state of mental erythrysm. The stage of brain fag then sets in rapidly. It begins with mental confusion and headache. The brain feels as though covered with a blanket that will not let ideas through. Ideas do not come readily and prolonged mental concentration becomes impossible. The mind wanders to other subjects and an effort to keep it concentrated upon any one thing causes a confusion of ideas. He cannot keep out other thoughts, while the main subject becomes dim and may be forgotten. This stage resembles senile impairment leading to dementia, but the psychasthenic can still evolve grand conceptions, while in senile impairment this is impossible as the ideas run along a lower plane. If the mental faculties are employed during the stage of brain fatigue or brain fag, and strong efforts are made to continue the mental labors, the stage of brain exhaustion is reached. In this condition thought is impossible and the patient is really in a state of mental collapse. During the second stage the will becomes weakened and cerebral impulses become slower and weaker and are conducted less forcibly by the nerves. The functional activity of the nerves becomes diminished but not from lowered functional capacity, as is the case in true neurasthenia. In the aged psychasthenia hastens the senile degeneration of the brain and it is a powerful factor in causing early senile dementia.

When psychasthenia and neurasthenia occur together they may be mistaken for general paresis. This disease is rare in old age, convulsions may appear and there is generally a feeling of exhilaration while in the other there is mental depression and phobias, instead of delusions of grandeur.

Treatment.—Mental rest is the most important factor in the treatment of psychasthenia. While in neurasthenia mental stimulation is indicated to dispel the depression, in psychasthenia physical exercise which does not required mental exertion must be employed. If there is at the same time mental depression, it will be necessary to resort to mental stimulants

like phosphorus, small doses of morphine, cannabis indica, etc., beside hygienic measures, such as change of environment, outdoor sports, preferably hunting and fishing with a cheerful companion. The use of aphrodisiacs recommended in the mental depression of neurasthenia is contraindicated in psychasthenia, since the latter is usually followed by senile dementia in which an abnormal recrudescence of sexual desires frequently occurs and gives rise to sexual perversions.

SENILE NEURASTHENIA

The term neurasthenia is applied loosely to a number of symptoms arising from constant and excessive brain and nerve fatigue. The term should be applied only to the condition of *nerve weakness* and not to the mental depression that accompanies it and is usually due to it, nor to the purely psychic phenomena of mental exhaustion which are described as *psychasthenia.* This psychasthenia is responsible for many symptoms that are also found in neurasthenia. Both conditions, neurasthenia and psychasthenia, frequently exist at the same time. Neurasthenia in the aged presents some peculiarities due to the generally diminished functional capacity and activity.

Etiology.—Neurasthenia is due to excessive nervous activity with insufficient repair and in addition probably to an autointoxication from the absorption of waste products arising from nerve activity. We find a similar condition when muscle is employed after fatigue sets in. A local toxemia makes further activity difficult and finally impossible. When this point is reached we get muscle exhaustion. In neurasthenia the point of complete nervous exhaustion is rarely reached as the mental depression and psychasthenia prevent further nervous activity as soon as nerve fatigue is felt. Under some extraordinary impulse the neurasthenic is able to exhibit some nervous energy, which would be impossible in complete exhaustion.

Nerve weakness generally follows prolonged excitement whether of business or pleasure, with improper recreation or insufficient exercise. It does not occur in those engaged in physical labors unless the character of the work necessitates frequent responses to sudden nerve impulses. The telegraphic operator waiting for orders which must be instantly transmitted,

the telephone switch board operator, the type writing operator, and all who must be on the alert for work requiring rapid action, are liable to neurasthenia. In the aged such nerve tension causes rapid nerve weakness and the symptoms appear early since the realization of advancing age is itself depressing, causing introspection and the recognition of failing powers. The sense or feeling of weakness is exaggerated, while under a proper stimulus the aged person will exhibit remarkable nervous energy. Predisposing factors are heredity, alcoholism, early excesses, worries and other causes of mental depression, disturbed circulation, toxemias and arteriosclerosis. The nervous or neurotic disposition in which there is excessive nervous irritability is the underlying factor found in most cases. It is very rare in females.

Symptoms.—In the aged the symptoms of neurasthenia are always accompanied by mental depression and the latter is frequently more marked than the nervous symptoms. In many cases the mental depression which ensues as the result of the recognition of the waning mental, physical and sexual powers, causes a diminution of will and energy and the aged person exaggerates his loss of power and nervous energy. This pseudoneurasthenia is a form of malingering. If there is a real neurasthenia present there will be the intention, but not the impulse to perform the intended act. The patient feels constantly tired and even the slightest task is performed under protest. Where he had been previously mentally alert and physically active he is now dull and apparently lazy. His movements are sluggish and are performed with an effort. A sudden danger will rouse him to activity but he soon relapses into a state of mental and physical depression. Neuroses of various organs are often found. Nervous dyspepsia is generally present, with anorexia, thirst, gastric and intestinal indigestion, constipation, while diarrhea occurs upon slight emotional excitement. Cardiac neuroses are frequently observed and vasomotor disturbances may occur. In spinal neurasthenia there is a feeling of weakness along the spine and tender points are found upon pressure along the spinal column. Other nervous symptoms occasionally observed are neuralgia, parasthesia, fine tremors, etc. Headache or hemicrania is sometimes present and various disorders of the special senses may

occur. In the senile cases it is often difficult to decide whether some of the symptoms are due to neurasthenia or to arteriosclerosis or to other senile changes. The mental symptoms are irritability, depression, introspection, phobias, etc. The aged patient watches his pulse and notes every change in frequency or rhythm, he observes his breathing, his skin, etc., indeed his mind is centered upon himself and the minutest change causes him to fear the worst. The morbid depression and fear of death lead to hypochondriasis which later resolves itself into a melancholia, this terminating in dementia.

Senile neurasthenia is a serious condition as there is generally a cerebral arteriosclerosis present which cannot be cured, its symptoms are persistent, and the mental state tends to melancholia and dementia. If psychasthenia coexists with neurasthenia the depression soon gives way to senile dementia.

Neurasthenia can be divided into four stages, an irritable period preceding the period of fatigue, the stage of nerve fatigue, the period following fatigue and preceding exhaustion and the stage of exhaustion. During the preliminary period the individual exhibits physical irritability. Like the man with his finger on the trigger waiting for the command to fire, the patient is ready to jump or start upon the slightest provocation. He does things rapidly when there is no necessity for speed, makes unnecessary movements and is in a state of nervous tension. In the stage of fatigue, he moves slowly and with deliberation and avoids unnecessary activity. In this stage mental depression appears and tends to inhibit motion. There is still some irritability with occasional outbursts of speed or exaggerated energy. If this is persisted in the stage of fatigue is followed by the intermediate period. Now it requires a sensible effort and a strong impulse to arouse nervous energy. In this stage the local neuroses appear, introspection becomes marked and we find the host of symptoms described. The stage of complete nervous exhaustion is rarely reached. In this stage there is complete loss of energy, motion and even eating becomes an effort. It may occur if under some powerful stimulus during the preceding period some extraordinary effort is made. This stage ends in collapse. There is no sharp dividing line between the stages, the first passing rapidly into the second, the second passing slowly into the third. The onset of the last may be

sudden. Aged persons generally seek medical aid when the stage of fatigue sets in; younger individuals make efforts to continue their work until this stage is well advanced.

Treatment.—The most important indication in the treatment of senile neurasthenia is to improve the mental condition. This can be done most effectively by the use of aphrodisiacs and a favorable result will have a more potent beneficial effect upon an aged man than any other stimulus. Change of scene, mental diversions, hobbies, out-door amusements, are all beneficial. There must, however, always be a cheerful companion to prevent a lapse into the habit of introspection. A day's fishing when fishing is good will rouse nervous energy and dispel mental depression, while quiet, rest, and the companionship of fellow sufferers in a sanitarium will *not* cure mental depression or neurasthenia. For the physical condition we can use strychnine, caffeine or arsenic, salt water baths and static electricity. Pleasurable excitement which will keep the mind occupied but will not confuse should form part of the routine treatment. There is probably nothing more effective to take the mind away from thoughts of the body than an old familiar air.

Insomnia, especially insufficient sleep at night, is not alone distressing; it invariably causes introspection with phobias. A hot bath or foot bath should be tried, hot malted milk taken before going to bed and if these fail we must give veronal or trional in 5- to 10-grain doses.

SENILE EPILEPSY

Senile epilepsy is a disease of old age only in so far as the ordinary epilepsy of earlier life may occur in the aged. Cases originating in old age present minor clinical differences, but as these differences are ascribed to a coexisting arteriosclerosis some authorities speak of it as arteriosclerotic or cardiovascular epilepsy.

Etiology.—While neither the etiology nor the pathology has been determined, in nearly every case one of the supposed causes of early life can be found. It may be an infectious disease, intoxication or autointoxication, powerful emotion, sexual excesses, traumatism or some cerebral disease. In some cases

no cause can be assigned and these are called idiopathic senile epilepsy. It is probable that in every case there is an irritation of some portion of the cerebral cortex which contains a focus left from some former cerebral disease. The irritation may come from blood toxins as in alcoholism, syphilis, nicotine or tuberculosis or from traumatism or a sudden emotion, etc. It is uncertain what the relation between senile epilepsy and arteriosclerosis is, but it is generally conceded that the vascular condition may produce the neurosis.

Symptoms.—Senile epilepsy possesses the pathognomonic element of suddenness. The first attack is generally as severe as later ones. The cry is frequently absent, but there are occasional premonitory symptoms, as headache, spasms, neuralgic pains, vertigo and frequently an aura. The convulsions are generally less severe and not as prolonged as in maturity. In other respects the convulsive seizure does not differ from that of maturity. The mind is, however, frequently affected and after several attacks dementia is liable to set in with progressive loss of mentality. It is hardly possible to mistake epilepsy for any other disease having convulsions. The convulsion of uremia is generally followed by coma and there is a history of renal disease. In apoplexy there is paralysis, meningitis is very rare in the aged and there is generally some paralysis, headache, mental disturbance, fever, irregular pupils, etc. Hysteria is rare in the aged and there is generally a history of daily attacks with emotional perversions, the attack ending in a flood of tears. In general paralysis, the disturbance of speech and the mental condition will serve to distinguish it.

Epileptiform convulsions can be produced by interfering with the cerebral circulation, as when compressing the carotids. In these cases there are clonic spasms but no aura, no cry, the sphincters are not relaxed, coma comes on gradually, there is no deep sleep after the attack and there is no mental impairment. In senile epilepsy the tonic spasm lasts but a moment, the clonic spasms are weaker, the legs are not thrown about as in younger life, and the sleep is less profound than in maturity.

Treatment.—The treatment is as in maturity. The underlying cause must be treated. If no cause can be found we must fall back upon the bromides, preferably the bromide of strontium

in 10-grain doses several times a day with total exclusion of meat. The nerve stimulants like strychnine are contraindicated.

Neuroses in the Aged

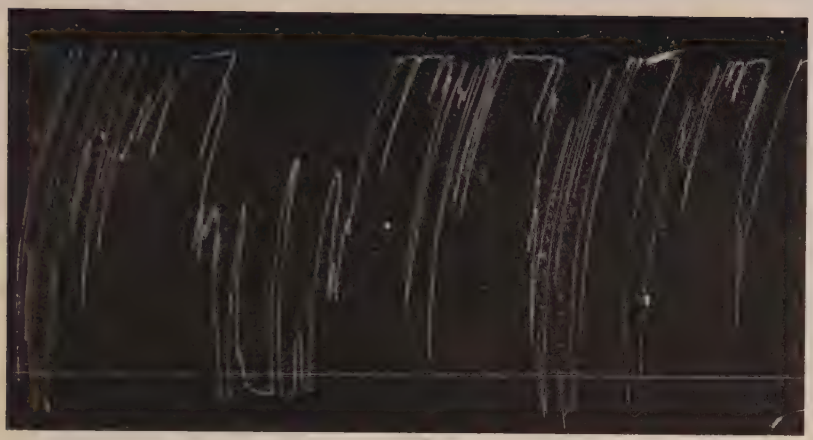
The aged frequently present nervous phenomena for which no cause or pathological change can be found.

(Some of these like epilepsy and neurasthenia present marked peculiarities and are placed among the modified diseases. Others like senile tremor and senile abasia are probably manifestations of senile changes of the cord and are classified accordingly, while under senile neuroses will be placed various neuralgias and other neuroses that are rare or unchanged.)

Hysteria is extremely rare in the aged and does not differ from the same disease in maturity. Cases may be carried over from earlier life or follow traumatism, the latter cause producing at times extreme depression and any of the numerous manifestations of hysteria. Large doses of bromides and narcotics may be required to quiet the patient. In giving narcotics in these cases they should be combined with respiratory and cardiac stimulants.

Hemicrania or migraine is of rare occurrence in the aged and is then usually carried over from maturity. It is really a disease which becomes milder and finally disappears with advancing years and when it does occur it is either a symptom of cerebral arteriosclerosis or a prodromal symptom of some serious cerebral or nervous disturbance. It may precede apoplexy or mental breakdown. When occurring as a prodromal symptom it is usually associated with irritability, anxiety, nausea, vertigo and other nervous phenomena. The treatment depends upon the cause. For the relief of the headache, a large dose of bromide of sodium should be taken and if the heart is in good condition this may be combined with one of the coal tar preparations like antipyrin or acetphenetidin with caffein or ammonium carbonate.

Chorea is rarely met with in the aged although choreiform convulsive movements of the hands are sometimes seen in the course of other nervous diseases especially in those of traumatic origin. Very few cases of the chronic Huntington's chorea have been reported and these were invariably associated with senile dementia. The ordinary chorea appears generally in a mild



Tremorgraph—Chorea. (Neustaedter, *Med. Record*, July 17, 1909.)

Tremorgraph—Epilepsy. (Neustaedter, *Med. Record*, July 17, 1909.)

Tremorgraph—Hysterical tremor. (Neustaedter, *Med. Record*, July 17, 1909.)

form, is sometimes limited to one side and the movements may be rhythmical. A one-sided chorea has been observed preceding a hemiplegia. The choreic movements are identical with the same movements seen in childhood. The treatment consists in the administration of arsenic in increasing doses until the physiological effects of the drug are produced. Chronic chorea is not benefited by arsenic or any other drug.

Diabetes insipidus is sometimes classed as a neurosis, although it is in almost every case a symptom of a nervous or cerebral affection. It occurs most frequently in connection with hysteria, epilepsy and neurasthenia, follows apoplexy or traumatic affections of the brain or cord and may be a temporary condition following some strong emotion. The aged sometimes complain that they pass an excessive amount of urine when suffering from dilatation of the bladder. They have then a frequent desire to urinate and pass a small amount each time but the total amount is not excessive.

The treatment of diabetes insipidus depends upon the causative disease.

INSOMNIA

The aged frequently complain of insomnia, although in most cases it is a pseudo-insomnia for which nothing need be done. They take frequent naps during the day and are then unable to sleep more than a few hours at night. Slight exercise induces fatigue and they fall asleep after their meals and after doing a little work. Mental exercise brings on brain fag and they fall asleep while reading the paper, listening to a lecture or sermon or after an argument or dispute. In this way the aged individual may get three or four hours sleep during the day and his night's rest being broken perhaps by an overdistended bladder, he complains of insomnia. To cure this pseudo-insomnia it would be necessary to prevent the daylight naps, which aside from being a harsh procedure, would interfere with recuperation and repair. The best that can be done in these cases is to draw off the urine and give a hot drink and a hot foot bath at night. They will fall asleep more readily and will not be disturbed by an irritable overfilled bladder, but it will not increase the total amount of sleep. Drugs are unnecessary in this condition.

Real insomnia may be due to pain, fever, toxemia, nervous or cerebral disease. There may be inability to fall asleep, broken sleep or insufficient sleep. The treatment depends upon the cause. If hypnotics are required they should be selected from the carbamide group, not from the methane or chloral group of hypnotics.

NEURALGIA

Various forms of neuralgia occur in the aged, trifacial neuralgia being the most frequent and the only one for which a conclusive etiological factor has been found. (This is described in the second group.)

Trifacial neuralgia may be due to other causes than compression of the terminal fibers in the bony substance of the lower jaw. In most cases it is secondary to another local disease as caries of the teeth, disease of the mucous membrane of the nose, maxilla, or frontal sinus, exostoses, or it may be secondary to an infectious disease, or it may be due to cold, local irritation, etc. In some cases no cause can be found. The differentiation of the affected branches of the trifacial nerve depends upon the location of the painful pressure points. In supraorbital neuralgia this is found in the supraorbital notch of the frontal bone, in supramaxillary neuralgia it is found in the infraorbital foramen, and in inframaxillary neuralgia the painful pressure point is at the mental foramen. These points are surrounded by painful areas. The pain is severe, there being usually a constant dull ache in the region of the affected nerve branch with paroxysms of agonizing sharp pain over the painful pressure points. In the treatment of trifacial neuralgia the cause should be determined and, if possible, removed. Supraorbital neuralgia may be due to disease of the frontal sinus, while many cases of supramaxillary and inframaxillary neuralgia are due to dental caries. Where the cause is unknown or cannot be removed the treatment must be directed to the relief of pain. The injection of 10 minims of a 4 per cent. solution of cocaine will generally give temporary relief. Relief and sometimes permanent cure is obtained by alcohol injections. Other local measures are morphia and atropia injections, the application of a piece of cotton soaked in ether, the ethyl chloride spray,

cocaine ointment, galvanism and heat. In extreme cases when local measures fail surgical interference including resection of the nerve branch may become necessary.

Occipital neuralgia is rare in the aged. It is generally due to sudden exposure to cold, sometimes to gout or arthritis deformans, rarely to infectious diseases. Painful pressure points are midway between the mastoid process and the first vertebra and at the posterior junction of the sternocleidomastoid muscle and the occipital bone, the pain shooting into the surrounding tissue and sometimes extending over the entire scalp. The neuralgic paroxysms are intense, lancinating and last but a moment. The treatment is purely local and consists of the application of moist heat, cocaine ointment, galvanism. It may be necessary to give morphine to secure sleep. Surgical intervention is rarely necessary.

Brachial neuralgia may occur in the aged as a result of disease of the heart, aorta or subclavian artery, of rheumatism, gout, infectious diseases, diabetes, cancer, hysteria or local disease. In most cases no etiological factor can be found and the disease is classed as a pure neurosis. There are usually several pressure points where branches emerge from their muscular folds. Beside the paroxysmal attacks there are often local disturbances such as paresthesias, herpes zoster, local hyperemia or anemia, these occasionally alternating. The treatment is as for occipital neuralgia.

Ischial neuralgia occurs occasionally in the aged and is usually due to sudden changes of temperature or prolonged standing, sometimes to pressure upon the nerve by growths, hardened feces, etc., sometimes again to local passive congestion, inflammations or other causes of neural irritation. Bilateral neuralgia may be due to a disease of the cord or to a constitutional disease. Painful pressure points are found all along the ischial nerve and the pain radiates but slightly. Slight at the beginning, the pain becomes rapidly more severe with occasional paroxysms of intense sharp pains lasting but a moment. Pressure and motion increases the pain, but the paroxysms occur frequently at night also. In lying, sitting and standing the patient assumes a posture which will shield the affected side from pressure and motion. The treatment is as for occipital neuralgia. In some cases a mixture of equal parts of chloral and camphor

pencilled along the nerve will give relief. Many of the drugs useful in neuralgias in earlier life cannot be used in the aged. Aconitin is dangerous, iodide of potassium is useless, arsenic and quinine are of doubtful value. Alcohol injections are occasionally of benefit, sometimes they produce a neuritis and local tissue inflammation. In many cases we must resort to morphine for temporary relief and surgical measures, such as nerve stretching or resection.

PREFERENTIAL DISEASES OF OLD AGE

The fourth group includes the diseases most frequently found after middle age although they may appear earlier. Some of these diseases are primary, as diabetes, gout, cancer. The prevalence of these diseases in late life and their infrequency in early life would seem to indicate some relation to the process of involution. The secondary diseases of this group include chronic diseases many of which arise in old age from a focus left over from an earlier acute disease.

CARCINOMA

Ignorance of the nature and pathogenesis of cancer makes it difficult or impossible to assign it to its proper group. It is here classed as a preferential disease owing to its prevalence in advanced life. If we consider it an infectious disease it would be properly placed among the infectious diseases of the fifth group. If we accept the view that it is a perversion of the normal process of involution of certain tissues we would be obliged to assign it to the first group. The latter seems to the author to be the most plausible explanation of its origin and nature and is in accord with the theory of tissue-cell evolution. Cohnheim's theory is that cancers arise from faulty embryonic development, embryonic cells remaining dormant until late in life. The tissue-cell evolution theory is based upon analogy with evolution in higher and more complex forms of life. Atavistic tendencies appear in all forms of higher life and in all stages of evolution. May not the primitive cells show the same tendencies? These tendencies would become more pronounced at that stage of evolution when the cells are departing from their

most perfect condition and their functions are no longer best fitted for the welfare of the economy. In senescence, functional activity of the cells is lessened, the organism becomes functionally weakened and the tissues are altered. If at this time there occurs a cell traumatism or nutritional perversion which interferes with the steady progressive cell evolution there will be a change in the character and properties of that cell. It may cause complete destruction of the cell, or further impairment of its functions or perverse stimulation and if there are atavistic tendencies there will be a return to an earlier type, or to cells of an earlier evolutionary period with disordered growth and disordered functional activity. Cancer never begins *en masse*, but in a single cell or in several adjoining cells possessing similar tendencies. Its further growth is by extension from a single focus and by the formation of new foci by means of cancer cells carried in the blood or lymph channels, these cells stimulating cells of other tissues to disordered growth or activity. Cancer is not a metastatic disease, *i.e.*, one that shifts its location away from the original site. Heredity is probably the most important etiological factor in the atavistic tendencies in cell life, just as it is in the life of the human being as a whole. It is impossible here to take up all forms of cancer, and all the localities in which they may appear, or to go into the pathology of cancer growths, therefore, little more than the symptoms and treatment of the most important forms will be considered here (the malignant growths of the skin being placed among the modified skin diseases). Syms points out a precancerous stage and shows that benign tumors, chronic ulcerations, chronic inflammations, scars, and prolonged irritation are prominent precursors of cancer. He quotes Young, who demonstrated an immense proportion of carcinomas among cases of enlarged prostate; Bloodgood, who studied sixty-five cases of pigmented mole which became malignant; and Mayo who found between 60 and 70 per cent. of gastric carcinomas on the sites of gastric ulcers. The recognition of this precancerous stage would save many cases from the later ravages of cancer. There is apparently an antagonism between cancer and the infectious diseases. Cancer is very rare in lepers or syphilitic cases or in malarial districts. A large percentage of cancer cases never had any infectious disease, while on the other hand erysipelas rarely develops in cancer

cases. Tuberculous cadavers show cancer in 4 per cent., non-tuberculous cadavers show cancer in 11 per cent. of cases.

There are certain clinical features common to all cancers. The most prominent is the cancer cachexia, a rapid emaciation, an anemia with rapid diminution in hemoglobin percentage and in the number of red cells and a muddy, sallow complexion, loss of strength keeping pace with the emaciation and mental depression. Primary cancers are generally followed by secondary ones, the most frequent location of secondary cancers being in the lymphatic glands, and where the primary cancer attacks an abdominal organ other than the liver, the liver is the usual seat of it. Pain is a frequent but not a constant feature. It is usually neuralgic in character, and due to pressure of the growth upon a nerve or ganglion. It is often more severe and persistent in the secondary cancer than in the primary lesion. In many locations the growth can be neither seen nor felt but as it increases in size it presses upon adjoining tissues and symptoms pointing to disease of such tissues appear. This is a late feature. Cancer growths near openings have a tendency to grow toward the opening occluding it, and when in channels or tubes, the tendency of growth is inward, causing stenosis with final complete occlusion. Cancer does not produce fever. When there is fever it is due to an accidental infection or inflammation.

There is no cure for cancer except complete extirpation of the growth by surgical means, before metastases have appeared. Even then the relief is often only temporary, as foci for future growths have usually been produced. The character of the operation will depend upon the findings after the growth is reached. Drug treatment can be only palliative. Jacobi strongly recommends methylene blue in doses of 1 grain gradually increased to 3 grains three times a day, combined with extract of belladonna, in inoperable carcinoma.

Oral Cancer.—Cancer of the mouth includes cancer of the lip, tongue, cheeks, tonsil and pharynx. They occur most frequently in tobacco smokers.

Cancer of the lip is usually a primary epithelioma having its seat at the junction of the mucous membrane and the skin. It begins in most cases as a papule or small, hard wart which may exist without change for months or years. It then begins

to itch or annoy and a crust forms. The patient picks this, leaving a slight ulceration which refuses to heal but increases in size and depth while the surrounding tissue becomes hard and swollen. In some cases the disease begins in a fissure or a pustule which later ulcerates. The further progress of the disease follows one of two courses. The ulcer may become larger and deeper until it destroys a large part of the lip or the lip may be filled with a mass of cancerous tissue forming a cauliflower-shaped hemorrhagic tumor, which will bleed upon the slightest irritation or will crack and become covered with foul ulcers. Later the adjoining or neighboring tissues become involved. The submaxillary glands are early affected, becoming hard, swollen and painful, while the other glands of the lower jaw and the neck are soon similarly involved. The viscera, however, are rarely affected. Cachexia is not marked until the disease is well advanced but it then progresses rapidly and may cause death from exhaustion. The only disease with which cancer of the lip is liable to be confounded is syphilis. The history, the presence of other syphilides and the Wassermann reaction will clear up this source of error. The only effective treatment is complete extirpation of the growth before glandular involvement. If the glands are affected these also must be removed. Delay is fatal.

Cancer of the Tongue and Mouth.—Cancer of the tongue is usually a primary epithelioma beginning as an indurated swelling at the surface of the organ. The swelling is painful upon pressure, neuralgic pains shoot sometimes through it, and the tongue can be protruded with difficulty only, while swallowing becomes painful. In some cases the swelling increases until the greater part of the tongue is involved, in others it ulcerates, the ulcer growing larger and deeper until the whole oral cavity is a foul-smelling ulcerated mass. The salivary glands swell and may ulcerate also. Cachexia sets in early and causes death from exhaustion in from three to twelve months. In some cases death is due to a deglutition pneumonia. The only treatment possible in these cases is surgical. Antiseptic mouth washes and local analgesic remedies may be required for temporary relief of the fetor and pain, and rectal alimentation may become necessary. The progress of the disease cannot be halted by medicinal

measures. In some cases the cancerous progress begins in the mucous membrane of the cheek as a small ulcer which spreads rapidly in all directions, soon involving the tongue. In rare cases the ulcer burrows through the cheek. The further progress is as in cancer of the tongue.

The above description applies to cancer of the tonsil also. A few cases of primary cancer of the parotid gland have been recorded. It begins as a swelling under the angle of the jaw, increasing rapidly in size, pressing in all directions and interfering with deglutition and occasionally with respiration. The tumor itself is not painful but pressure upon nerves causes intense pain. It may be mistaken for parotitis but its constant growth without fever or pain upon pressure will serve to distinguish them. Surgical interference is the only remedy.

Cancer of the Larynx.—This is usually a primary epithelial growth; sometimes it occurs as an extension of a carcinoma from an adjoining tissue, rarely as a secondary cancer. Its favorite seat is upon the vocal cords, though it may occur elsewhere. It begins as a surface infiltration which forms first excrescences then ulcerations which extend in size and depth. It resembles cancer of the rectum in its slow development, slow progress, slight cachexia and late involvement of the lymphatics. The early symptom of cancer of the vocal cords is a persistent hoarseness without pain or cough. The laryngoscope shows a broad-based growth which may be smooth or uneven, slightly reddened and with an infiltrated area around it or at one side. The motion of the affected band during respiration and phonation is greatly impaired, thereby differing from benign growths in which the motility is not altered. Cancer growths in other parts of the larynx will produce symptoms of stenosis or pressure according to the direction in which the growth extends. It may cause difficult deglutition or difficult respiration or pain on motion, these symptoms increasing until deglutition or respiration becomes impossible. When ulceration occurs there is a mucopurulent discharge, later, a fetid odor of the breath shows necrotic changes. Secondary cancers of adjoining tissues frequently follow and their symptoms may be more severe than the symptoms of the primary growth.

Treatment is early surgical intervention.

Cancer of the Lung.—Cancer of the lung is very rare and most of the reported cases were secondary cancers in which the primary one was of greater clinical importance. Primary cancer occurs only by extension of a carcinoma of the finer bronchial tubes along the bronchioles and alveoli into the lung tissue. The tissue first becomes hard then breaks down in the center while the borders extend. Cavities may thus be formed, simulating tuberculosis. During the period of hardening the physical signs resemble pneumonia. Secondary pulmonary carcinoma is usually multiple and very small, simulating miliary tuberculosis. The symptoms are not clear and it is often difficult to say whether it is cancer, local tuberculosis, pneumonia, pleurisy, miliary tuberculosis or bronchiectasis. There is no fever, rarely pain, but occasionally hemorrhage or a hemorrhagic expectoration, the blood being intimately mixed with mucus. Dyspnea with shallow breathing is a constant symptom. In rare cases the tumor will cause bulging of the chest wall, or displacement of the heart.

Treatment is entirely symptomatic. Operative procedures have been reported but none have ever been successful in the aged. Arsenic has been used with temporary success in the cachexia and morphine is generally the only means of relieving the distressing symptoms.

Pleural Cancer.—Cancer growths in the pleura are rare and almost always secondary. The early symptoms are those of pleurisy, later pressure symptoms and pain occur. The cachexia is marked, as the disease in the pleura is usually a late affection. A rare form of primary endothelial cancer called *lymphangitis carcinomatodes* is peculiar to the pleura. Secondary growths occur in the lymph channels and may invade the lungs and bronchi. The early symptoms are those of serofibrinous pleurisy without change of the border of dulness upon change of position. Puncture produces a serochylous exudate containing epithelial débris and round, generally polynucleated cells. The needle passes through denser tissue than in ordinary pleurisy (Schwalbe). The growth is frequently painful, the pain radiating toward the arm. The pressure upon the lung will produce dyspnea and if erosion of blood-vessels occurs there will be a hemorrhagic expectoration. This form of malignant growth progresses more slowly than other forms of cancer, the cachexia

sets in late and the fatal issue may not be reached until several years after the initial symptoms. There is no curative treatment for pleural cancer. Aspiration may relieve dyspnea if there is much exudate and narcotics must be given toward the end to relieve pain.

Mediastinal Cancer.—Cancer in the mediastinum is often a primary lymphadenoma of the mediastinal lymphatics, occasionally secondary to cancer in a neighboring tissue. The symptoms are principally due to pressure upon organs or to displacement of tissues by the growth. Pain is infrequent but there is generally tenderness over the site of the growth. The diagnosis must be often made by the pressure symptoms and by exclusion. Cachexia sets in early and death is due to either exhaustion or asphyxia from pressure upon the trachea or bronchus. The treatment is surgical.

Esophageal Cancer.—Esophageal cancer is usually an epithelioma, occurring in the lower third of the tube. In the aged the growth generally proceeds upward and into the cavity of the tube, narrowing and finally completely occluding the caliber. The symptoms are a progressive dysphagia with a sensation that the food is stopped at a certain point and powerful efforts at deglutition must be made to carry it past the obstruction. Another symptom is the cachexia. The growth rarely proceeds outward and therefore symptoms of pressure upon other tissues are rare. There is occasional pain, never severe, at the point of obstruction. Food when regurgitated is covered with mucus, sometimes with blood. The neighboring lymphatics are sometimes involved but death from asthenia generally sets in before secondary cancers appear. Esophageal sounds may be used to determine the location of the obstruction, but any effort to force the sound past that point may cause inflammation or perforation and collapse. Perforation through the cancer growth is rare however. The only treatment is early operation. In the meantime foods must be given in liquid form until complete stenosis has occurred after which rectal feeding must be resorted to. Narcotics and cocaine are only of temporary benefit to relieve pain but the pain in the aged is seldom severe enough to require treatment.

Gastric Carcinoma

Gastric carcinoma is the most frequent of the visceral cancers occurring in the aged. It is usually a cylinder-celled epithelioma. The soft encephaloid and the hard scirrhus cancers are occasionally found, but the colloid form is rare. Writers generally agree that the pylorus is the favored site of gastric cancer, but there is considerable difference of opinion as to the main etiological factor. Wilson and McCarty of the Mayo clinic found that 71 per cent. had developed on the base of an old ulcer; French says a history of ulcer or injury is obtained in 6 per cent. but fully half of the cases operated upon show evidences of previous ulcer; while Weinstein agrees that some cancers do develop from ulcers, but he rejects the high percentage given by Wilson and McCarty. Ewald points out the rarity of gastric ulcer in the aged and sees in traumatism affecting the walls of the stomach a notable etiological factor. To harmonize these diverse views with the prevalence of cancer at the pylorus we must believe that most gastric cancers in the aged result from the scar of an early latent ulcer. Gastric carcinoma is generally a primary cancer and is followed by involvement of the lymphatic glands, often by growths in the liver or gall-bladder, occasionally in the peritoneum, intestines or other tissues. These secondary cancers may produce more disturbance than the primary lesion.

Symptoms.—There are no early pathognomonic symptoms of the disease. The rarity of ulcer in the aged disposes of this precancerous stage, but when an ulcer exists sudden or rapid diminution of free hydrochloric acid and the presence of lactic acid, in conjunction with other symptoms, points strongly to cancer and is nearly pathognomonic. Weinstein says a sudden abrupt onset in a person who had been in perfect health is one of the strongest links in the cancer chain, while Cabot declares that any type of dyspepsia occurring in a person over forty who had had no such trouble before, is strongly suggestive of cancer. These statements do not hold good in senile cases, for in most cases there is a history of gastric disturbance going back perhaps for months before there are any other symptoms of cancer, and gastric disturbances of the aged are rather frequent, yet few develop into cancer.

The early symptoms depend mainly upon the location of the growth. There is generally loss of appetite and a rapidly developing cachexia. If the cancer is situated at the fundus, pain, nausea and vomiting occur late and the disease progresses more slowly than when it is situated at the pylorus. The cachexia is associated with a pronounced anemia, the red cells may sink to 3,000,000 or less while the hemoglobin may drop to 50 per cent. or less. Emaciation sets in early. It proceeds rapidly if the cancer is at the pylorus where it interferes with the passage of food into the duodenum or if situated at the cardiac orifice where it interferes with the passage of food into the stomach. If either orifice is completely occluded death from starvation soon follows. The skin in cancer cachexia presents a muddy, sallow or ocherous hue most pronounced on exposed surfaces, and it is usually dry and wrinkled. The secondary group of symptoms—pain, nausea, vomiting—may vary in degree or be absent altogether. Pain usually comes on soon after the ingestion of food, but it may occur paroxysmally at any time. It is an early symptom in cases where a cancer develops upon an existing ulcer and a late one if the cancer is at the fundus. The degree of pain varies. Nausea and vomiting are usually early symptoms although in the aged vomiting is infrequent and requires severe straining. The vomited matter consists of food in various stages of digestion, mixed with mucus and sometimes blood. Food vomited two or three hours after a meal is usually foul or sour smelling. Blood is generally present early but in quantities so small that a microscopic or chemical examination may be required to determine its presence. The "coffee ground" vomit, which contains digested blood with the hemoglobin converted into hematin, is a late but almost pathognomonic sign of cancer. Lactic acid bacilli are generally found in the vomited matter. Of the physical signs the most important is the presence of a growth, usually at the pylorus, firm, smooth or nodular, and generally movable. It rises and falls with respiration and in the aged, who generally have wasted abdominal muscles, it can be grasped during expiration. A growth at the fundus, usually found at the lesser curvature is not palpable. The involvement of the lymphatics confirms the diagnosis of cancer in doubtful cases. While there is not a single pathognomonic sign of early gastric cancer (except the very rare occurrence of

cancer cells in the vomitus) and each individual sign and symptom may be found in some other condition, there are almost invariably several symptoms which taken collectively are conclusive of cancer or serve to exclude other conditions. The diseases which might be mistaken for gastric cancer are ulcer, chronic gastritis, benign growths, cancers outside of the stomach, and pylorospasm. The discovery of dissolved albumin in the stomach contents an hour after taking the Ewald test meal is pathognomonic of advanced gastric cancer. Benign growths in the stomach are very rare, and the tumor of pylorospasm will usually disappear. Ulcer and gastritis can be excluded by the history and the examination of the stomach contents.

Treatment.—The certainty of a fatal issue without operation and the possibility of a cure or at least the prolongation of life by operation justifies operative procedure in every case, however hopeless the outlook may be. Drugs, except for the relief of distressing symptoms, are useless. In senile cases especially, early operation is imperative and drug treatment may not even relieve symptoms unless given in toxic doses. The only indications for drug treatment are to relieve the pain, nausea and vomiting, while awaiting the operation. The most effectual drug for these symptoms is cocaine in 1/8-grain doses. Morphine and other opiates still further diminish the motility of the organ, prevent peristalsis and weaken the respiratory centers. Theoretically food given with acidulated pepsin and predigested foods ought to be beneficial; usually, however, they are of small service, as little is absorbed from the stomach, and where a pyloric stenosis exists little if any finds its way into the duodenum. The main advantage derived from these foods is less likelihood of fermentation. If the vomited matter smells sour resorcin, salol or the sulphocarbolates can be given. Hemoglobin, manganese and arsenic may be administered to improve the anemia, although they are rarely of much service. If the cancer is at the cardiac orifice drugs and food must be given in liquid form. Rectal alimentation is of service for a few days, but it is impossible to introduce sufficient food that way to completely nourish an individual.

Intestinal Cancer.—Nearly two-thirds of all intestinal cancers occur in the rectum. Sutton says that 75 per cent.

occur in the rectum and 25 per cent. occur in other parts of the large intestines. Rectal cancers are, as a rule, mild in their symptoms, progress slowly and cause comparatively little disturbance until far advanced. Secondary cancers occur late and the cachexia is rarely as pronounced as in cancers elsewhere. In many cases the symptoms of pressure upon adjoining tissues are more marked than the other symptoms and signs of a growth. Pressure upon a nerve or plexus will produce neuralgic pains, while pressure upon a vein will cause varix or edema. There are no pathognomonic symptoms of a non-palpable cancer. A cancer in the sigmoid or rectum can usually be seen through a colonoscope and a rectal cancer can usually be felt, by digital examination, as a hard, ulcerating mass. Growths in other parts of the intestines can sometimes be felt through the flaccid walls but it is necessary to eliminate first tumors of the liver, kidney, stomach and spleen.

The early symptoms of intestinal cancer are irregular stools, sometimes constipation, at other times diarrhea, sometimes hard, at other times soft or watery stools, sometimes copious then again scanty, and almost always containing traces of blood. Mummery says small frequent urgent stools indicate rectal cancer. If the cancer is above the cecum the blood is mixed with the stool, if below the cecum it covers the stool. There is flatulence and borborygmus, sometimes colicky pain but more often an ache in the region of the lesion with a painful spot over the growth. The diagnosis, however, is never assured until the growth is palpable. Other diseases liable to be mistaken for intestinal cancer in which no tumor can be felt are chronic ulcerative enteritis, syphilis, tuberculosis, concretions and actinomycosis. The last one is very rare, while syphilis and tuberculosis can be determined by serum tests. Concretions will disappear under a brisk cathartic and chronic ulcerative enteritis has diarrhea with pus and shreds of mucus in the feces. As the cancer increases it diminishes the caliber of the bowel and finally causes complete stenosis with the symptom of intestinal occlusion. The treatment is surgical. In no other form of cancer is the surgical prognosis as favorable as in rectal cancer. Without operation the prognosis is fatal. The only drug indications are for the relief of pain and constipation. If the opera-

tion is delayed until secondary cancers form or until complete occlusion has occurred, recovery is doubtful.

Cancer of Liver.—Hepatic cancer is almost always secondary to gastric cancer, cancer in other parts of the digestive tract, or cancer of the female genitals, and occasionally to cancers of some other part of the body. Less than 5 per cent. are primary. A fairly pathognomonic symptom-complex is enlargement and tumefaction of the organ, presence of a tumor which is painful on pressure, colicky pains about the organ, radiating toward the right axilla, and the general cachexia with pronounced jaundice. In some cases nodules can be felt at the edge or upon the surface of the liver. The jaundice is of hepatogenous origin, noticeable on the conjunctiva and increasing as the growth interferes more and more with the secretion of bile. Owing to bile retention and interference with its passage to the gall-bladder and intestines, another set of symptoms is produced. These are, anorexia, especially a distaste for meat and fat; flatulence, meteorism, clayey, foul-smelling stools, dark brown urine containing bile pigment, intense pruritus, and, usually, nervous and cerebral symptoms. In some cases rapid emaciation with jaundice and a dull ache on pressure are the only suggestive symptoms. Primary cancer does not always give these symptoms. In cases in which the carcinoma is in the substance of the liver and does not reach the surface, the organ will be increased in size but no nodules will be felt and icterus may be slight or even absent if the growth does not obstruct the free flow of bile. While there is no single pathognomonic sign, yet the history of a primary cancer, the rapid emaciation, jaundice, pain and increased size of the organ will suffice to exclude most other diseases. Cirrhosis and syphilis are infrequent in the aged. It is sometimes difficult to differentiate between a cancer of the liver and that of an adjoining organ in the aged, as the liver then usually lies lower in the abdominal cavity than in maturity and growths in adjoining organs may become adherent to the liver. It is, however, only in the cases where the primary disease is so mild that it is overlooked that a mistake can be made. Cachexia is common to all types. Cachexia without marked jaundice points to cancer in some other organ than the liver, *i.e.*, gall-bladder, or ducts, or pancreas. It is impossible to differentiate between cancer of the liver and that of the gall-

bladder unless there is a palpable tumor which can be defined. In cancer of the pancreas the pain is to the left of the median line, there is often occlusion of the pylorus with dilatation of the stomach and if the tumor is palpable it will be found that it does not move with respiration. Glycosuria may also be present.

There is no known cure for cancer of the liver and death generally occurs within a few months after its symptoms appear. If secondary to another cancer, operation can serve no purpose whatever. In the rare cases of primary cancer of the liver, benefit from an operation is possible although such operations are almost invariably fatal. All that can be done is to temporarily relieve distressing symptoms by narcotics, analgesics, hypnotics, etc.

Cancer of the Gall-badder.—With the exception of the location of the growth, the symptoms of cancer of the gall-bladder are the same as those of cancer of the liver. A palpable enlargement of the gall-bladder, because of its position under the liver, appears as an enlargement of or growth upon the liver itself. The disease is almost always secondary and what has been said of the prognosis and treatment of cancer of the liver applies to this condition. Gall-stones may give similar symptoms but the history, paroxysmal colic, possible finding of stones in the stools on the one hand, and the history of a primary cancer or lymphatic involvement and rapidly progressive cachexia, will suffice to differentiate between them.

Cancer of the gall-ducts cannot be differentiated from hepatic or biliary cancer.

Cancer of the Pancreas.—Cancer of the pancreas is rare, but most cases occur in advanced age. The clinical picture described by Bard and Pic includes icterus, progressive and without remissions, enormous distention of the gall-bladder, readily perceived upon palpation, no increase in the size of the liver, temperature habitually subnormal, rapid emaciation and cachexia, short duration of the disease, sometimes a tumor in the epigastrium, absolute decoloration of the fecal matter, abundant biliary pigment in the urine and frequent albuminuria. In the absence of tumor there symptoms apply as well to cancer of the gall-bladder. DaCosta found a tumor in 13 out of 137 cases of cancer of the pancreas. More frequent symptoms are pain in the region of the pancreas, the symptoms

of pyloric or duodenal stenosis, fatty stools and glycosuria. While the diagnosis of a cancer is not difficult in the presence of rapidly progressive cachexia and pain, in the absence of a tumor it is often impossible to determine whether the cancer is in the pancreas, duodenum or gall-duct. Cancer in the body or tail of the pancreas is very rare and when it occurs it does not produce jaundice, as the growth doer not compress the bile-duct. There is, however, pain, constant or paroxysmal, not aggravated by food, radiating to the left and of more intensity than in any other abdominal tumor. Packard points out an area of tenderness above and to the left of the umbilicus indicative of pancreatic disease and this with the cachexia and other symptoms of cancer is sufficiently suggestive to make a fairly certain diagnosis. The only treatment is surgical. Symptomatic remedies may be employed for the relief of pain and to supply medicinally the deficient pancreatic juice and bile.

Prostatic Cancer.—Prostatic cancer is one of the more frequent forms of cancer in the aged. It is usually a primary scirrhus, occurring either as a cancer growth upon an enlarged prostate or as a hypertrophied prostate which became cancerous. Fuller says 1/7 or 1/8 of cases coming under his observation with symptoms of prostatic obstruction were cancer. An early symptom of a primary cancer is an increased frequency of urination and rapid symptoms of obstruction which may appear in from four to six weeks, later, involvement of adjoining tissues and cachexia. In some cases there are no early symptoms except those of hypertrophy, which may exist for years before the obstruction is sufficiently marked to give decided symptoms and these symptoms proceed slowly. Hematuria, the blood appearing at the end of urination, points to cancer or acute cystitis, but when associated with prostatic obstruction it is strongly suggestive of cancer of the prostate. Digital rectal examination reveals a hypertrophied prostate usually nodular, irregular, hard and when adjoining tissues are involved it is immovable or there is a sensation under the finger as if adjoining tissue is being dragged along. Pain, except the dull ache that accompanies hypertrophy, does not occur until adjacent tissues are involved and then it is frequently a sharp pain radiating from the groin down the thighs, and toward the sacrum, occasionally to the suprapubic region. When the

active symptoms, frequent urination, rapidly increasing prostatic obstruction with hypertrophy, blood at the end of urination, with rapid emaciation and other concomitants of cachexia appear, a mistake is hardly possible. The only successful treatment of prostatic cancer is early extirpation of the gland. If the disease has invaded adjoining tissues, there is no likelihood that any operation can radically free the patient of his trouble (Fuller). The increasing obstruction to urination will sooner or later make operative interference imperative. Drug treatment is useless except for the relief of pain, when the narcotics may be given.

Cancer of the Bladder.—Cancer of the bladder occurs occasionally in the aged male and less frequently in the aged female. It originates usually in a benign papillary fibroma which may have existed for years. Secondary cancers generally follow uterine cancer in the female and cancer of the rectum or prostate in the male. There are no pathognomonic symptoms of cancer of the bladder unless particles of the growth are found in the urine. The usual symptoms, pain, hematuria and dysuria, may occur in other conditions, notably in benign tumors and in acute cystitis, while the symptoms of some cases of cancer are mild and intermittent. If above-mentioned symptoms appear in the course of a cancer of the rectum, prostate or uterus it is strongly suggestive of secondary cancer of the bladder. A positive diagnosis, however, requires the use of the cystoscope or the frequent examination of the urine for cancer cells. The tumor is rarely large enough or located so favorably that it can be felt. In primary cases cachexia appears late.

If the disease is primary and localized surgical measures may effect a cure. If secondary cancers have occurred, operation is useless except possibly to relieve an occlusion of the sphincter. Bangs reports several cases of inoperable cancer in which daily irrigation of the bladder with a hot solution (100° raised slowly to 105° F.) of 1/2 per cent. creolin relieved the irritability, lessened the hemorrhage and diminished the size of the growth. The effect was not permanent but relief was secured for several months.

Cancer of the Testicle.—Cancer of the testicle in the aged is almost always secondary to cancer of the prostate, bladder or rectum, and is then readily diagnosed by its rapid increase in

size. It is not painful except on pressure. When occurring as a secondary cancer the neighboring lymphatics are already infiltrated and operation is useless. In the rare cases of primary cancer in the aged the disease may remain quiescent for years, providing the testes have been removed early. The danger of delay lies in the involvement of the ileolumbar lymphatics and extension through them. If these glands have not been affected complete cure is possible.

Cancer of the Scrotum.—Cancer of the scrotum usually occurs as a papillomatous growth on the site of a scar, wart or eczema and follows the usual course of skin epithelioma. The treatment is excision. If performed before the lymphatics are involved complete cure is possible.

Cancer of the Penis.—Cancer of the penis may occur as a primary or secondary disease. The usual location is upon the glans where it begins as a painless wart which rapidly increases, forming a cauliflower excrescence. It rarely invades the corpus cavernosa, but the inguinal and retroperitoneal glands are involved early. In those cancers which involve the skin, as in cancer of the penis and scrotum, early operation generally affects a cure, providing the neighboring glands are not involved, or if involved, are completely removed. Treatment with the X-ray, radium, etc., is still experimental and while justifiable they often fail and valuable time is lost thereby.

Cancer of the Female Genitals.—In considering cancers in the aged we must include cancers of the female genital organs, although these belong exclusively in the domain of the gynecologist. The most frequent of these is cancer of the uterus. This generally begins as a primary benign neoplasm which becomes malignant. A provisional diagnosis can usually be made if, after the menopause, a hemorrhage or a serosanguineous discharge occurs, or if a persistent watery leucorrhea becomes blood tinged or assumes a fetid odor. Neuralgic pains occur if a nerve is pressed upon; usually, however, there is little pain, but a constant dull ache in the lumbar region. Violet says that an early diagnosis can usually be made by introducing a sound and gently moving it over the inner surface of the uterus. If there is cancer the physician can feel the sound scrape over the roughened surface of the growth. To make the diagnosis absolute, curettement and microscopic examination of the scrapings

are necessary. Cachexia sets in early but is not well marked until the disease is well advanced. Montgomery points out that, where there is a history of previous tubal inflammation a menorrhagia and watery discharge indicates cancer of the Fallopian tube. Cervical cancer can usually be seen through a speculum and is easily felt. Moulton says, however, that when a cervical cancer has reached the stage when it can be diagnosed unhesitatingly by the touch, eye, or history, then it has reached the border line between possibility and impossibility of cure. The early treatment is solely surgical, and delay occasioned by the use of medical measures simply lessens the chances of successful operation. Cancer of other parts of the female genitals is rare in the aged. When one occurs it is usually an epithelioma following a surface lesion such as eczema, excoriations from irritating discharges, scars of chancres or chancroids, etc. The treatment is excision of the mass but recurrence either on the site of the original growth or in the neighboring inguinal glands is of frequent occurrence.

Cancer of the Breast.—This is usually a primary cancer, occurring in most cases soon after the menopause. According to Isaacs from 80 to 90 per cent. of all tumors of the breast are malignant and of the remainder a large proportion will become malignant if permitted to progress. The early symptoms are the presence of a hard mass, pain or tenderness and diminished mobility of the breast with elevation of the nipple of the affected side. A bloody discharge from the nipple is strongly suggestive of carcinoma. Later symptoms are retraction of the nipple, adhesion of the growth to the chest wall, lymphatic involvement, edema of the arm, ulceration, and cachexia. The treatment is surgical; the earlier performed the better the prognosis. Abbe succeeded in healing an ulcerating inoperable cancer of the breast by radium.

GRAWITZ'S CACHEXIA

Etiology.—This disease, described by Grawitz as a "fatal cachexia without discernible anatomical cause," somewhat resembles pernicious anemia, but the red blood cells show no degenerative change, though they may be diminished in number. No change in any organ or tissue has been found to explain the rapid anemia, emaciation, loss of strength and general physical

breakdown, and it is assumed that the disease is due to some deleterious substance which has gained access to the blood; possibly the chromaffine substance of the adrenals.

Symptoms.—The symptoms are rapidly increasing pallor, emaciation, loss of strength, and a mental and physical depression, leading to fatal exhaustion.

The differential diagnosis between this disease and pernicious anemia rests upon the examination of the blood. The red cells number from 1,000,000 to 3,000,000 and they show no degenerative change. The disease resembles the cancer cachexia and can be distinguished from cancer only by the local and secondary symptoms of the latter disease.

Treatment.—There is no known remedy. It is usually treated as pernicious anemia, and is always fatal.

CHRONIC LARYNGITIS

Chronic Laryngitis occurs either as a localized disease or a part of a more extensive chronic inflammation of the nares, pharynx, bronchi, etc. It appears in two forms, a hypertrophic catarrh corresponding to chronic hypertrophic bronchitis, and an atrophic catarrh corresponding to the senile atrophic bronchitis.

Etiology.—The hypertrophic form of chronic laryngitis occurs most frequently in speakers, singers, and others who use the voice excessively, or in those who have had repeated attacks of acute laryngitis, or from extension of a chronic catarrh into adjoining tissues and lastly after infectious diseases in which the mucous membranes are profoundly involved, as in influenza and tuberculosis. The atrophic form occurs from the same causes that produce senile atrophic bronchitis. This form of laryngitis is a true senile disease, depending upon the presence of an atrophied mucous membrane which had been irritated.

Symptoms.—In the hypertrophic form there is hoarseness, especially in the morning until the secretion which has collected during the night has been expelled. Involvement of adjacent tissues, as thickening or paresis of the vocal cords, may cause complete aphonia. There is a persistent feeling of tickling or irritation, rarely pain, a desire to cough but no relief after coughing, occasional dysphagia and dyspnea. In the atrophic form the voice is squeaky and weak but there is no hoarseness

or aphonia, the throat feels dry but there are occasional spasmodic attacks of coughing with expectoration of a tenacious gray mucus. If the mucus accumulates upon the cords or controlling muscles there will be hoarseness, perhaps aphonia, but with the removal of the secretion the voice is again thin and squeaky. The diagnosis can readily be established by the laryngoscope. The grave forms of laryngitis associated with tuberculosis, cancer or syphilis can generally be differentiated by the history and accompanying symptoms of the primary disease.

Treatment.—In the atrophic form of chronic laryngitis local stimulation is required and can best be accomplished by the inhalation of hot water to which menthol and eucalyptol have been added. If the expectoration is purulent, oil of turpentine inhalations should be used instead. If the mucus is thick and scanty the syrup of the hypophosphite of ammonium should be given in dram doses several times a day. In the hypertrophic form local application of mild astringents is indicated, such as blowing dry astringent powders or brushing the inflamed area with a weak solution of iodine or nitrate of silver.

Hygienic measures and prophylaxis are self-evident.

CHRONIC HYPERTROPHIC BRONCHIAL CATARRH

This is the old man's winter cough, the most frequent bronchial affection of the aged, and bronchiectasis is almost always associated with it.

Etiology.—This form of bronchitis comes on with the advent of cold weather and is due to the alternate chilling and warming of the bronchial mucous membrane, from the difference in temperature between the inspired and expired air. It does not appear when the patient spends the winter in a warm equable climate. The disease occurs most frequently among those who have been exposed for years to such deleterious influences as dust, vapors, rapid temperature changes, etc., and, in almost every case, a tendency to catarrhal affections, carried over from earlier life, can be established. It is occasionally found in cases where excessive cautiousness against temperature changes and other causes of bronchitis has produced a condition of great sensitiveness in the bronchial mucous membrane. In

Lung, Emphysema and Bronchiectasis. (Natural size.) (From Coplin's "Manual of Pathology.) *A*. Emphysema. vesicle. *B*. Enlarged peribronchial gland. *C*. Enlarged peribronchial gland, pigmented. *D, D, D*. Dilated bronchi.

such, a slight indiscretion as a draught, or a momentary chilling of the surface, produces a bronchitis.

Pathology.—There is a passive hyperemia with thickening of the mucous membrane and enlargement of the mucous glands. The latter are usually open and are surrounded by dark-colored zones. The blood-vessels are filled and tortuous. Numerous small elevations and depressions are found in the mucous walls and the tissue feels soft and velvety. Later on, if there have been violent fits of coughing weak spots in the bronchial walls will result which dilate, producing *bronchiectasis*. These dilatations form sacs and pouches and may become cystic reservoirs of mucus or of muco-purulent matter. In some cases there are alternating areas of dilatation and stenosis, the latter caused by hypertrophy of the mucous membrane or by hyperplasia of fibrous connective tissue. The mucous membrane of these dilatations is sometimes atrophied and leathery and under severe strain of coughing it may rupture permitting the contents to enter the lung tissue.

Symptoms.—Chronic hypertrophic bronchial catarrh begins with a slight cough which gives little distress and is followed two or three days later by an abundant expectoration. The expectoration is muco-purulent, thick, yellow, sometimes tinged with green. A brownish expectoration, if specially abundant in the morning and associated with a spasmodic cough, indicates bronchiectasis. In some cases of chronic hypertrophic bronchitis the expectoration becomes purulent, thin and grayish, or heavy and greenish, and has a fetid odor. This may come from abscess or gangrene of the lung, tuberculosis, or long retention in a bronchiectatic reservoir where the mucus became purulent. In the last case there are no severe constitutional symptoms, but if due to abscess or gangrene there are symptoms of septic infection. The physical signs are as in simple bronchitis. Large dilatations gave amphoric breathing, cavernous resonance, possibly pectoriloquy and large bubbling râles. The physical signs of bronchiectasis are not clear but the diagnosis can usually be made by the large amount of secretion brought up in the morning with a spasmodic cough while, if there is no complicating bronchiectasis, the amount brought up in the morning exceeds but by little the amount expectorated at other times of the day. The symptoms of bronchiectasis persist

during the whole year while the symptoms of hypertrophic bronchitis disappear with the advent of warm weather to reappear at the next approach of winter.

Treatment.—The successful treatment of chronic hypertrophic bronchial catarrh depends upon the ability of the patient to go to a warm equable dry climate, but not at a high elevation, and remain there all winter. The disease will probably not appear while he is there but if he returns to a cold climate, the disease will return also. As long as he is obliged to breathe cold air he will suffer, and medicinal treatment is only palliative as long as the causative factor remains. Little can be done by internal medication, but the inhalation of creosote, terebene or eucalyptol is sometimes of service. Expectorants useful in acute bronchitis are generally contraindicated in this disease. The occasional administration of 1/100 grain of atropine may diminish the secretion and if the secretion is tenaceous the muriate of ammonia with syrup of senega may be given. If there is a putrid bronchitis a 2 per cent. spray or inhalation of phenol may be used to destroy the fetor but the treatment must be directed to the causative condition. Hygienic measures, such as warm clothing, freedom from draughts, feet protected from dampness, etc., are necessary adjuncts to the treatment.

PULMONARY EDEMA

Pulmonary edema occurs frequently in the aged as a secondary terminal disease, appearing in some cases during the death struggle. More often it initiates the series of phenomena that are associated with the process of dissolution. There is a transudation of serum from the blood-vessels into the interstitial tissue and air vesicles, blocking the latter and preventing aeration of blood. A recurrent type has been described but in the aged the first attack is almost always fatal.

Etiology.—In most cases, in the aged, it follows a passive hyperemia, either a hypostatic congestion due to long confinement to one position, or an obstruction to the return circulation due to cardiac or pericardial disease. In these cases separation of the serum occurs during stasis and it passes out of the vessels. Pulmonary edema also occurs in hydremic conditions as in

nephritis, cirrhosis of the liver, anemia, scurvy, etc., in which diseases there is a tendency of the serum to ooze out through the vessels. In some inflammatory conditions, as in pneumonia, bronchiolitis, etc., there may be stasis with separation of the serum from the blood. In extreme debility with weakened circulation, and in many fatal diseases, there is, toward the end, a relaxation of the blood-vessels which permits the exudation of serum and its transudation into the surrounding tissues and into the air vesicles.

Pathology.—The affected portion of the lung becomes lighter in color and heavier in weight, it pits upon pressure and upon opening the chest the lung does not collapse. Serum is found in the alveoli and interstitial tissue and if there has been a pulmonary congestion the serum is blood streaked. On sectioning, the serum exudes.

Symptoms.—The first symptom of pulmonary edema is usually a sudden, severe, or a rapidly increasing, dyspnea which is soon followed by an abundant, frothy mucus which may be blood streaked. The respiration is increased in frequency and the patient makes violent efforts to get air, sitting up and bringing into play all the respiratory muscles. There is inspiratory and expiratory dyspnea, the expiration being accompanied by an audible rattle as the air bubbles up through the tubes that are occluded by serum. Cyanosis often occurs toward the end. Some cases die of asphyxiation with the symptoms of choking, in others there is coma. The physical signs are dulness over the site of the edema, bubbling râles heard with inspiration and at the beginning of expiration, feeble respiratory murmur. The sudden or rapid onset and the history of an antecedent causative disease distinguish it from other pulmonary diseases.

Prognosis.—Pulmonary edema in the aged is almost invariably fatal, death usually occurring in from one to twenty-four hours after the onset of the disease. It occasionally makes its appearance during the last few minutes before death.

Treatment.—In a disease which is almost always rapidly fatal we are justified in employing any measures which might prolong life. The usual remedies, wet or dry cups, hot fomentations, mustard and turpentine applications, are useless in the aged. The time for their use was during the passive hyperemia when they might have equalized the circulation by producing

a superficial hyperemia, but after transudation of serum has taken place they cannot stimulate absorption. In hydremic states the theoretical treatment is to secure rapid elimination of fluid by means of hydragogue cathartics, diuretics and diaphoretics. Such rapid elimination, however active, weakens the heart and does not remove the serum from the air vesicles. Venesection will either immediately destroy the patient or will afford but temporary relief by relieving the local congestion. In such cases there may be a temporary absorption of the transudate, but the vessels soon fill again and the serum will again transude into the vesicles. The report that a case was relieved by turning the patient on his stomach, placing him across the bed and supporting his abdomen with a high bolster, while his head was hanging over the edge, thereby allowing the serum to flow out by gravity, induced the author to try this in one case. The patient suffocated. Oxygen inhalation will relieve the cyanosis for a time, but as the disease progresses and greater areas of lung tissue are involved, the blood becomes more contaminated, finally the oxygen cannot aerate the blood sufficiently and coma and death ensue.

Suprarenal preparations will sometimes control and prevent further transudation if given at the onset of the disease, but they do not produce reabsorption of the transudate present in the air vesicles. If there is but a small quantity, it may be expectorated and the patient tided over by oxygen inhalations. The adrenal preparations are powerful vasoconstrictors and if there is cerebral arteriosclerosis they may produce cerebral apoplexy, but the gravity of pulmonary edema in the aged overbalances the possibility of producing apoplexy. The rapidly acting eliminants are powerful cardiac depressants and, if given, they must be combined with rapidly acting cardiac stimulants, preferably strychnine and digitalin. This applies especially to the drastic hydragogue cathartics.

PULMONARY GANGRENE

Etiology.—Gangrenous destruction of lung tissue results from the action of putrefactive bacteria upon diseased tissue. It is always a secondary disease, most frequently following an aspiration or deglutition pneumonia, or other form of pneumonia,

fetid bronchitis or bronchiectasis, cancer or tuberculosis. It may, however, occur in any pulmonary affection or may originate from extraneous sources of infection such as a perforating esophageal cancer or empyema, degenerating bronchial glands or traumas. A non-putrid gangrene occasionally occurs in the course of diabetes. A pulmonary embolus is also frequently followed by gangrene.

Pathology.—Pulmonary gangrene may be acute or chronic, circumscribed or diffuse, single or multiple. The tissue becomes first jelly-like, then softens into a pultaceous gray or greenish mass of fetid matter that contains shreds of tissue which had not undergone complete destruction. In the diffuse form this mass extends into adjoining healthy tissue, while in the circumscribed form the mass is limited by a growth of connective tissue. Where an opening into a bronchial tube exists, the mass is expectorated and the cavity may become completely cleared. An opening into the pleural cavity may cause a pyopneumothorax. There is usually an acute or a fetid bronchitis and often a pleurisy or empyema associated with pulmonary gangrene.

Symptoms.—This disease in the aged is usually acute and begins with active symptoms of septic infection, such as irregular fever, perspiration, and prostration. The severity of the symptoms depends upon the extent of the gangrene. If it is a small, single, localized area, symptoms will be mild, while in an extensive diffused gangrene there will be high fever, rapid prostration and emaciation, finally cerebral symptoms of delirium and coma appear and death soon results. An early, and sometimes the first, symptom of pulmonary gangrene is cough with fetid expectoration. The sputum is thin, greenish or dark gray or brown and contains, beside mucus and pus, bits of gangrenous tissue, Dittrich's plugs, crystals of fatty acids and under the microscope it is seen to be loaded with bacteria. The odor of the sputum is the decomposition odor of nitrogenous matter similar to the odor of decaying meat. This will often suffice to distinguish gangrene from abscess of the lung and bronchiectasis, in which the odor is due to fatty acids and resembles old cheese. There are sometimes traces of blood, and if a blood-vessel becomes necrotic there will be a hemorrhage.

Pulmonary gangrene in the aged is almost invariably fatal. Even in mild cases where the disease is localized and involves

only a small area, as when a small foreign body has been aspirated, there is always the danger of diffusion or the formation of secondary foci through aspiration of particles of putrid matter from the bronchi. Metastatic abscesses and gangrene may occur.

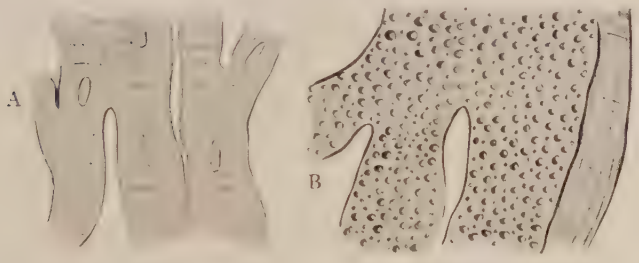
Treatment.—Only in rare instances will medical measures avail, while operative procedure is likewise rarely successful. The medical measures are the inhalation of disinfectants, such as creosote, guaiacol, turpentine, etc., and the internal administration of powerful expectorants as the syrup of the hypophosphite of ammonium (contraindicated in tuberculosis), syrup of senega or ipecac and also creosote, guaiacol and similar drugs.

The strength must be maintained by tonics, concentrated foods, and small quantities of alcohol, while incidental symptoms, as fever, pain, insomnia, etc., must receive appropriate treatment. It is possible that serum therapy holds a cure for this disease but at present the only chance for recovery, slight though it be, lies in operation.

Pyopneumothorax.—Pneumothorax, hydropneumothorax and pyopneumothorax are rare complications of pulmonary gangrene and occur when a gangrene or abscess opens into the pleural cavity. These diseases may occur from any cause which produces an opening into the cavity from without, or from the lungs, pleural or abdominal cavity. They are, therefore, liable to occur as a result of surgical operations, empyema, tuberculosis, abscess, or gangrene of the lung, sudden inspiratory effort causing rupture of alveoli into the cavity and abscesses of the abdominal cavity opening through the diaphragm into the pleural cavity, etc. Pneumothorax and hydropneumothorax are extremely rare, and when they occur they soon become infected. The symptoms which usually set in suddenly are pain and a sensation of tearing in the lung, dyspnea, sometimes cyanosis and anxiety, and occasionally expectoration of pus. The physical signs are the same as in maturity, the auscultatory signs being especially well marked in later life. The distention of the affected side is not marked owing to the rigidity of the chest walls, but intercostal distention is obliterated.

A cavity of a pulmonary tuberculosis may give similar physical signs but not the same symptoms, and is very rare in old

Fatty degeneration of heart, with thickened aortic leaflets and mitral stenosis (wooden wedge in button-hole opening), about one-half size. (Satterthwaite, *Medical Record*, May 14, 1910.



Fatty degeneration of cardiac muscle. *a* beginning changes; *b*, complete deegeneration, × 250. Satterthwaite, *Medical Record*, May 14, 1910.

age. Treatment is usually fruitless, as the causative condition is generally of a fatal nature. Aspiration may give temporary relief.

PULMONARY ABSCESS

Etiology.—Abscess of the lung occurs occasionally in old people, most frequently as a secondary complication of pneumonia. In influenza the bacilli sometimes cause minute abscesses in the lung, and the aspiration of purulent matter from the nose, throat or bronchi or of particles of food may also occasion it. Less frequent causes are perforation of the lung from an empyema, bronchiectasis or other pus cavity, or from without as from bullet wounds and other trauma, pyemia with the formation of metastatic abscesses, and tuberculosis.

Symptoms.—The symptoms in the aged are generally vague, although there are numerous symptoms and signs pointing to pulmonary disease and sometimes to septic infection. Where the disease follows or complicates an infectious disease, the earliest symptom is a purulent expectoration. The pus in the sputum is mixed with mucus and does not form coin-shaped plaques as in purulent bronchitis, nor lumps as in tuberculosis. The pus cavities in the senile are often little more than distended slits in the tissues, seldom rounded cavities such as appear in tuberculosis. The slit-like cavities are usually found in the lower lobe, the round cavities in the upper lobe where they simulate tuberculosis and in most cases a bacteriological examination is then necessary to determine whether it is a tubercular abscess or not. The physical signs of pulmonary abscess in the upper lobe are the same as in tuberculosis. The tympanic percussion note, prolonged expiration, large and fine moist râles and cough with expectoration which persists day and night, all point to a cavity which is constantly being emptied. When the abscess is in the lower lobe the pus collects during night and necessitates prolonged coughing and expectoration in the morning, with but little cough or expectoration during the rest of the day.

When the abscess is due to an aspiration or deglutition pneumonia there is little or no elevation of temperature but cachexia with anemia and emaciation soon sets in. The sputum

becomes purulent and shows on standing the characteristic layers of purulent expectoration. The heavy grayish lower layer contains bacteria, leucocytes, fatty acids, and elastic fibers. This layer has a foul, and in an old abscess, a fetid necrotic odor. The middle layer is grayish and watery and if a blood-vessel has been involved the liquid will be colored red or brown. The upper layer is mucus and contains air cells. The sputum of bronchiectasis is similar, and a deep-seated bronchiectasis in the lower lobe often gives signs similar to the physical signs of abscess. In these cases the more rapid development and the cachexia point to abscess, but the history may be necessary to determine the diagnosis.

Treatment.—The only certain method of emptying a pus cavity in the lung is by operative procedure, aspiration, or by resection of ribs and then aspiration. The uncertainty of the exact situation of the abscess makes rib resection the better course, but the method must be left to the surgeon. If the local and constitutional symptoms are mild, medicinal measures can be employed to favor emptying of the pus cavity by expectoration. This can sometimes be accomplished through the inhalation of guaiacol or creosote, the internal administration of expectorants as muriate of ammonia, syrup of senega and ipecac, and a posture which will permit the free flow of the sputum toward the mouth. This can be produced by raising the foot of the bed. The use of hygienic regulations, fresh air, concentrated food, tonics, etc., is self-evident.

CARDIAC HYPERTROPHY

Simple hypertrophy is normal in the aged, the increasing hyperplasia keeping pace with the increase in the resistance of the vessels, caused by arteriosclerosis. In the physiological hypertrophy the left ventricle alone is involved but there is no increase in the size of the cavity. When the walls of any of the other cavities become hypertrophied or dilatation sets in we have a pathological condition to deal with.

Etiology.—Cardiac hypertrophy is the result of excessive work, the muscle tissue increasing as all striped muscles increase in volume, when actively employed.

The principal causes for left ventricular hypertrophy are

increased arterial resistance as in arteriosclerosis, nephritis, or as a result of vasoconstrictor drugs; or toxic irritation as in uremia, goiter, gout, and infectious diseases; cardiac defects as valvular disease or myocarditis, or pericardial adhesions; finally excessive or prolonged exercise as in athletic sports.

Right ventricular hypertrophy occurs when there is some obstruction to the circulation in the lungs, as in emphysema, or when there is mitral disease present. Pericardial adhesions may cause excessive work for the ventricular muscle and produce hypertrophy.

Auricular hypertrophy is invariably accompanied by dilatation and is due to valvular disease.

Pathology.—The heart is increased in size, it is rounder and less pointed than in its normal state. If the hypertrophy is confined to the left ventricle, the heart is pear shaped, but if both ventricles are involved it is longer and oval. The walls may be increased to twice their normal size, the increase being hyperplastic (numerical hypertrophy).

Symptoms.—In the simple hypertrophy of old age associated with arteriosclerosis there are no symptoms referable to the heart as long as the heart's action is not disturbed. Exercise or excitement will produce palpitation and may produce cerebral hyperemia with headache, vertigo and tinnitus. At such times the face becomes flushed, there may be a nervous twitching and psychic manifestations, such as irritability of temper, may appear. Similar symptoms are produced when there is cardiac irritation from a distended stomach. In the hypertrophy associated with other cardiac lesions the symptoms of the latter mask the symptoms of the hypertrophy.

The physical signs in left ventricular hypertrophy are an increased area of impulse and visible cardiac pulsation, an increased area of the apex beat to the left of and below its normal position, percussion dulness is increased to the left and downward, and the heart sounds are increased in intensity, the first sound being dull and prolonged.

In right ventricular hypertrophy there is epigastric pulsation, the apex beat is displaced to the right and downward, percussion dulness is increased to the right of the sternum, and the second sound is heard loudest over the pulmonic orifice. In auricular hypertrophy and dilatation the percussion area is increased up-

ward. Except in simple hypertrophy of the left ventricle the symptoms and signs are modified by the accompanying lesions and if there is dilatation present, all of these may be altered except the area of percussion dulness. Hypertrophy is differentiated from dilatation by the regularity and strength of the heart action, by the more intense second sound, absence of murmurs and absence of symptoms referable to pulmonary engorgement.

With increasing arterial resistance there is an increasing hypertrophy to a point where the limit of functional capacity is reached. Beyond this point compensation is broken, dilatation ensues and the further progress is the history of cardiac dilatation.

Treatment.—The main indication in the treatment of simple hypertrophy is to maintain compensation and to defer as long as possible the inevitable break when the limit of the functional capacity is reached. If there is an underlying pathological condition this must receive attention. Hygienic measures must be taken to prevent intense or prolonged mental and physical strain, excesses or fatigue. Heart stimulants are contraindicated and only in case of palpitation following excessive exercise or in case of fever are cardiac sedatives like aconite or veratrum permissible. Alcohol must be strictly forbidden.

CARDIAC DILATATION

Cardiac dilatation is an increase in the size of the cavities. It is invariably a secondary condition either following a hypertrophy which has reached the limit of its functional capacity or some other condition which has impaired the tonicity of the heart muscle. There is a limit to the working capacity of muscle fibers, but prolonged or excessive work produces fatigue before this limit is reached. Further activity can be aroused only under a forced stimulus, until exhaustion sets in with complete inability to work under any stimulus. Fatigue demands rest and exhaustion compels rest, during which recuperation and repair take place. Every contraction of the heart is followed by a short refractory period during which it cannot respond to stimulation. This corresponds to the rest period following exhaustion. With the increasing arterial resistance, due to arterio-

sclerosis, the work of the heart is increased and this is made possible by an increase in the number of muscle fibers or hyperplasia of tissue. Gradually the work increases more rapidly than the increase in tissue can keep pace with, while the rest period is not prolonged proportionately and the muscle finally reaches the point of the limit of its functional capacity. Then, being unable to respond to further increased activity, the fibers begin to degenerate, lose their tonicity, stretch and cannot contract fully. Thus is then produced a distention of the walls with dilatation of the chambers. In some cases this is brought about by the weakening of the walls through malnutrition. In other cases the internal tension is greatly increased and the tonicity of the muscle fibers is thus impaired. This may occur during prolonged strain and it may occur rapidly or even suddenly upon any violent and sudden exertion. In most cases, however, the inception of the dilatation arises from increased internal tension as soon as the limit of the functional activity of the heart muscle is reached.

Etiology.—There are three general causes for cardiac dilatation: (1) exceeding the limit of the functional capacity of the heart, (2) degeneration of the heart and (3) increased internal pressure. The first is the usual cause in senile cases, and the method of production has been explained. The second, *i.e.*, degeneration of the heart, may be due to the process of involution, to toxins, especially that of pneumonia, to typhoid fever, influenza, erysipelas and other diseases accompanied by pyrexia, or to malnutrition. In these cases there is usually no change in the thickness of the walls. The third, or increased internal pressure, may occur secondarily to cardiac weakness with impaired circulation, the auricles becoming overdistended while the ventricles are unable to completely empty themselves. This may occur in myocardial degeneration, valvular disease or arteriosclerosis. Increased internal pressure may also be produced by the ingestion of large quantities of fluids. The so-called "Münchner Bier Herz" is a cardiac dilatation due to this cause. Sudden or prolonged exertion as in violent athletic sports may produce a rapid dilatation from increased internal pressure. This is the usual cause of death of contestants just after a supreme effort on the athletic field. In these cases the walls of the heart are generally thin.

Pathology.—The cardiac walls may be normal, hypertrophied or thin. In senile cases there is usually hypertrophy and all chambers are dilated. In other cases the right ventricle is first dilated followed by the left auricle, right auricle and lastly the left ventricle. There is, however, no uniformity in the order in which the chambers become dilated and in some cases of myocardial degeneration the dilatation is localized over the seat of the degeneration. The muscle fibers show degenerative changes and frequently the nerve ganglia are altered. The venæ cavæ are generally dilated if the right auricle is dilated. Dilatation is generally associated with valvular disease and the valves show the well-known changes in structure and anatomical relations.

Symptoms.—The symptoms vary with the form of dilatation. If there is a cardiac hypertrophy there may be a progressive dilatation without any symptoms, or with symptoms masked or counterbalanced by the symptoms of hypertrophy until dilatation becomes more marked or until a sudden strain produces a sudden or rapidly increasing dilatation. When occurring suddenly or rapidly there is a severe pain in the cardiac region, dyspnea and weak, rapid or irregular heart action. If coming on slowly there is but a gradual weakening of heart action and but occasional shortness of breath. These symptoms may be present for months before they are sufficiently severe to attract the attention of the patient. In cases not accompanied by hypertrophy, the symptoms appear more rapidly and are more pronounced from the beginning. In the form especially in which the walls are thinner than normal, the early symptoms are quite marked. In these cases there is palpitation, arrhythmia, in which the rate is irregular and the force diminished, the pulmonary circulation becomes impeded and there is incomplete aeration of blood and dyspnea on slight exertion, the arteries are incompletely filled and the veins are distended. As the dilatation increases there is a constant palpitation or a feeling of throbbing and irregular wobbling of the heart, the dyspnea becomes permanent, the face is pale, the lips cyanotic and on excitement the entire face becomes livid or cyanotic. The pulse is irregular and weak. Irritability results, followed later by diminished mental power. When the disease is far advanced, the symptoms become more marked, there is cyanosis, scanty albuminous urine, edema, occasional syncope, later the liver, spleen, kidneys and stomach

become involved through defective circulation and impaired venous return and their functions are disturbed. Dropsy may increase until there is general anasarca, but in the aged, death usually occurs from pulmonary edema before the dropsy is far advanced.

The principal physical signs are the increased area of dulness with short, feeble heart sounds. There is often arrhythmia, either the galloping arrhythmia or embryocardia. There is an indistinct and increased area of cardiac impulse with a diffused and weak apex beat. In thin persons a wavy movement over the precardial space can be seen and felt. Occasionally a thrill can be felt and if the right heart is affected the jugulars are prominent and dilated. As dilatation is usually associated with valvular disease there are also the symptoms and signs of the respective valvular lesion. In senile cases the pulse is of no service in determining the diagnosis. It is usually weak, irregular and intermittent but when the radial artery is sclerosed it may be hard, while beats may be lost in transmission from the heart to the wrist.

Diagnosis.—Observation of the physical signs ought to differentiate dilatation from hypertrophy in which the impulse, apex beat and heart sounds are strong and clearly defined. In thoracic aneurysm and mediastinal tumors the area of dulness is upward. In pericarditis with effusion the area of dulness, friction sounds, regular rhythm, and absence of vesicular murmur in the parts of the lung covered by the effusion will clear up the diagnosis.

Prognosis.—The prognosis in senile cases is bad. The disease is incurable and sometimes rapidly fatal. If it follows an aortic insufficiency or stenosis it usually runs a regular course; *i.e.*, relative mitral insufficiency, dilatation of the left auricle, pulmonary engorgement, hypertrophy and dilatation of the right ventricle, then of the right auricle, with general venous engorgement. This is the order of cardiac involvement but it may proceed rapidly or slowly or one or more stages may proceed more rapidly than others. Dilatation following diffuse myocardial degeneration proceeds rapidly and there is no order in the valvular involvement. Sometimes pulmonary engorgement evidenced by dyspnea and cyanosis occur soon after the initial symptoms of degeneration appear. Life can usually be

prolonged with care and treatment, but death will result from cardiac exhaustion, pulmonary edema or from degeneration of some other organ. Treatment is the same as that of valvular lesions.

FATTY DEGENERATION OF THE HEART

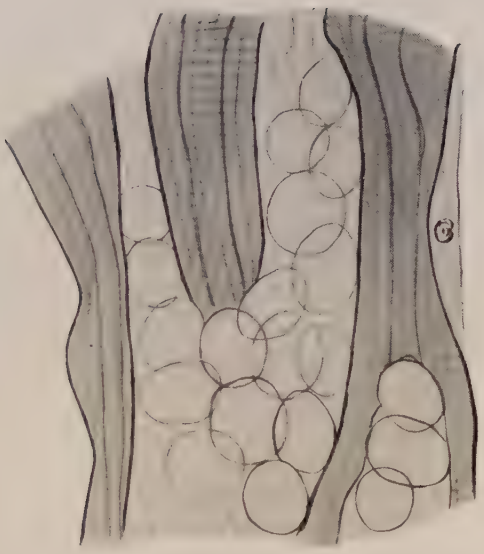
Fatty degeneration when occurring in the aged is generally due to impaired nutrition through sclerosis, embolism or thrombosis of the coronaries. It may follow fatty infiltration or occur in the progress of cancer. It is not, however, a true senile degeneration. It is assumed that where fatty infiltration exists, fat makes its way into the muscle cells. In other cases protoplasmic activity is impaired and instead of reproducing protoplasm it produces a suboxidation product, fat. According to this view fatty degeneration is secondary to a primary degeneration due to impaired nutrition.

Etiology.—Fatty degeneration may occur at any period of life and the same causes to which it may be due in earlier life may produce it in old age. The predisposing causes are, insufficient nutrition as from coronary sclerosis or from the general malnutrition of old age, from perverted nutrition as in general obesity and from fatty infiltration. These may also be the exciting causes. It may also occur in the course of infectious diseases, cachexias, Bright's disease, chronic alcoholism, phosphorus and arsenic poisoning, pericarditis, cardiac hypertrophy, etc.

Pathology.—If the degeneration is general, the heart may be enlarged, pale or yellowish and flabby, resembling fatty infiltration, or it may show little or no change. In many cases the degeneration is localized, involving the walls of a single chamber or of a still smaller area.

The muscle fibers lose their nuclei and striations, become granular and the sarcolemma is filled with granules. When the disease is far advanced the whole muscle cell seems to be filled with fat granules and oil globules. The disease rarely proceeds to that degree in the aged, as the whole heart is usually involved in these cases and owing to the general senile impairment of the organ, dilatation and its consequences follow early.

Symptoms.—There are no early symptoms indicating fatty degeneration and there may be no change in the function of



Abnormal deposition of fat, ×845. (Satterthwaite, *Medical Record*, May 14, 1910.)

the heart until dilatation sets in. As the disease progresses there are usually symptoms of defective heart power, rapid exhaustion and palpitation or arrhythmia upon exercise, dyspnea, giddiness, vertigo and syncope due to cerebral anemia, feeble pulse and apex beat, and upon auscultation the heart sounds are found to be feeble and the first sound almost inaudible. The respiration is generally feeble and sighing with occasional attacks of cardiac asthma and in extreme cases the Cheyne-Stokes type of respiration may appear. Irritability of temper, probably due to cerebral anemia—a prominent symptom in all cardiac affections producing cardiac weakness—is an early symptom of fatty degeneration. An increased area of percussion dulness before dilatation may be due to hypertrophy or fatty infiltration.

After dilatation has set in the symptoms are referable to that condition. Death may result from sudden strain or emotion.

Treatment.—The treatment of fatty degeneration is symptomatic and hygienic before dilatation takes place, the object being to prevent failing compensation. Mental and physical exertion must be avoided, especially sudden strains and emotions. High altitudes are injurious. Alcohol and tobacco are interdicted, liquids should not be taken with foods and the food should be easily digestible for prompt assimilation. Constipation should be guarded against. Mild exercise is permitted but the patient must constantly bear in mind that a sudden strain may cause death. The Oertel terrain treatment and the Schott and Nauheim treatments are dangerous in old age. Digitalis is contraindicated, but in an emergency, when lost compensation has brought about a critical condition with irregular, rapid, weak heart and dyspnea, and there is danger of cardiac exhaustion, and strychnia, carbonate of ammonia, aromatic spirits of ammonia and other cardiac stimulants have failed, we are justified in giving hypodermically digitalin combined with nitroglycerin in doses of 1/100 grain each.

FATTY INFILTRATION OF THE HEART

Fatty infiltration is not a degeneration nor a senile disease, although it occurs most frequently in obese persons after middle age.

Etiology.—In most cases it is part of the general process of suboxidation which causes obesity and is due to the same metabolic disturbance. It occurs mostly in women past the menopause and in men after the critical period which occurs about the fiftieth year. The underlying cause of obesity which frequently appears about this time is unknown.

Pathology.—Fat deposits around the heart, between the auricles, in the auriculo-ventricular groove, about the right ventricle and between the bundles of muscle. The heart appears larger and is pale or yellow and flabby. In advanced cases there is usually fatty degeneration and dilatation.

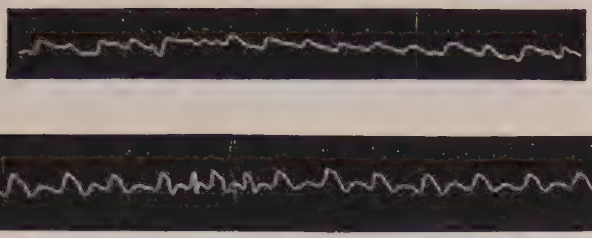
Symptoms.—There are no early signs or symptoms. It may be suspected when with increasing obesity there is dyspnea upon exertion and percussion reveals an increasing area of dulness. With the increase of fat about and in the heart, the heart muscle finds it more difficult to do its work, the heart becomes weakened and dilatation ensues. If fatty degeneration sets in, the dilatation proceeds more rapidly and the symptoms of that disease prevail.

Treatment.—The treatment of fatty infiltration is the treatment of general obesity. When degeneration or dilatation sets in, the treatment must be directed to the new condition.

VALVULAR LESIONS

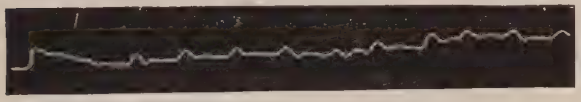
Valvular defect of some kind is found in most senile hearts, but they give no subjective symptoms as long as a compensatory hypertrophy exists. When compensation is broken and dilatation sets in, the symptoms become marked and the secondary diseases due to impaired circulation and venous engorgement quickly follow. The most frequent defects are aortic insufficiency, aortic stenosis and mitral insufficiency. Uncomplicated mitral stenosis is rare as the principal causative factor, acute endocarditis following rheumatism, infectious disease or toxin rarely occurs in old age.

The underlying cause of stenosis in old age is atheroma, either originating in the valve itself or extending to the valve from senile endocarditis or aortic atheroma. Insufficiency may be due to atheroma, cardiac dilatation or myocardial disease. There are, however, cases of aortic insufficiency in which the valve itself

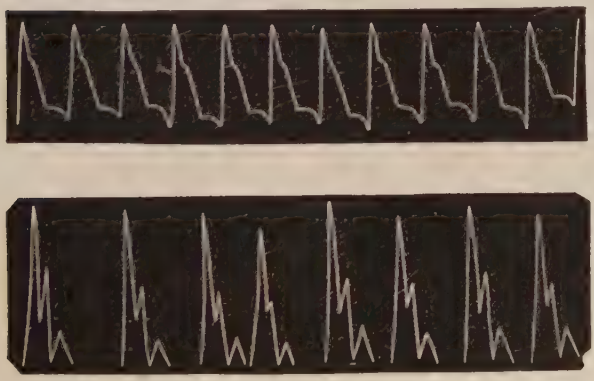


Tracing of Pulse in Mitral Stenosis. (From Tyson and Fussell's "Practice of Medicine.")

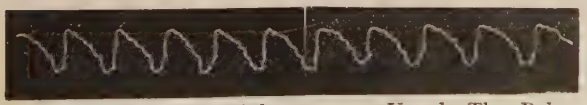
Pulse-tracing of Aortic Stenosis. (From Tyson and Fussell's "Practice of Medicine.")



Tracing of Pulse of Mitral Insufficiency. (From Tyson and Fussell's "Practice of Medicine.")



Tracings of Pulse of Aortic Regurgitation. (From Tyson and Fussell's Practice of Medicine.")



Sphygmogram of an Atheromatous Vessel—The Pulsus Tardus. (From Tyson's "Practice of Medicine.")

is not diseased but in which, owing to a dilatation of the aorta, the aortic ring is overstretched and cannot close completely. Preble has reported a similar relative insufficiency of the pulmonary valves. As a result of atheroma and degenerative changes in its structure, a valve may be both thickened and contracted producing insufficiency and stenosis at the same time. In some cases the mitral and aortic valves are involved, the mitral involvement being usually secondary to the aortic disease.

As long as the hypertrophied heart can compensate for the impairment of the circulation caused by diseased valves and sclerosed vessels, there may be little or no disturbance in the circulation and no marked symptoms of valvular disease, though the physical signs may be pronounced. When decompensation sets in, the symptoms of cardiac dilatation prevail, modified or accentuated by the symptoms of the valvular lesion. Patients having valvular disease in early life rarely reach old age and the valvular diseases found in the aged usually originate at that period of life.

The diagnosis of valvular disease in the aged is not difficult if there is but a single valvular defect. If there are two or more defects a discerning ear may be able to distinguish the separate murmurs, but other physical signs and the symptoms may be too complicated to be interpretable. A disturbing factor in these cases is the atheromatous aorta which adds its own train of symptoms. Where there is arrhythmia with a variable cardiac impulse the carotid pulsation will serve to determine the systolic sound. A frequent source of error in diagnosing valvular lesions is the altered position of the heart. In the aged the second sound is heard loudest in the third intercostal space and if the heart is greatly atrophied it may be most intense in the fourth intercostal space.

If degenerative changes originate in the valves, they begin by a thickening of the edge which becomes opaque, firm and inelastic. In the aortic valve the degenerative change begins in the corpus Arantii, and in the mitral valve at the margin of the leaflets. The endothelial covering undergoes the same changes as that of the arteries including ulceration and deposit of fatty and calcareous plaques. The valves may be thickened and contracted, appearing like misshapen pedicles which diminish the size of the orifice but cannot fully close it. The distorted

valves may take different forms and produce various shaped orifices, increasing or diminishing the size of the openings, partially blocking them or permitting any degree of regurgitation. Calcification is a late degenerative change. When this occurs decompensation rapidly ensues and the impairment to the circulation caused by the weakened heart produces rapid degeneration in other organs and tissues.

Before taking up the valvular lesions in detail a few diagnostic points applicable to senile cases will be given as follows.

A murmur or abnormal sound is heard when the blood is sent against a resistance at the orifice or when, in the rebound, the blood returns to a cavity through an incompletely closed orifice, or if the valves are roughened. In the first case the valve does not open completely and there is an obstruction or stenosis. This is due to a thickened or distorted valve or to a growth at the orifice. In the second case the valve does not close completely and we get a regurgitation or as it is usually called incompetence or insufficiency. This may be due to a defect of the valve or it may be due to a dilatation of the cavity or of the aorta, whereby the orifice is stretched. The diagnosis of a valvular lesion is made primarily by the location of the murmur and the time of its occurrence in the cardiac cycle. The obstructive murmurs are heard loudest over the valve, the regurgitant murmurs are heard loudest back of the valve in the cavity into which the blood rebounds, the former are carried forward in the direction of the flow, while the latter are carried in the direction of the rebound. We can thus localize mitral murmurs about the apex, aortic murmurs near the base to the right or left of the sternum and tricuspid murmurs behind the middle of the sternum. Pulmonic murmurs and tricuspid stenosis are extremely rare, and occur only when, through loss of compensation and myocardial degeneration, all the valves break down. In point of time the aortic stenosis occurs during the systolic contraction, while aortic regurgitation occurs after the blood has left the heart and a small quantity is forced back by the contraction of the aorta. This occurs during diastole The auricles contracting immediately before the ventricles, a mitral or tricuspid obstruction sound would occur therefore immediately before the systole or in the presystolic period. A mitral or a tricuspid insufficiency permits the return of blood from the ventricle during the con-

traction of that chamber, therefore the murmurs of these lesions occur during the systolic period. Abrahams has shown how the valvular lesions affect the pulse when the arm is held in different positions. In aortic regurgitation, if the arm is raised in a vertical position, the jerking, collapsing pulse is felt at an early stage of the disease and this variety of pulse remains throughout this disease. The pulse in aortic stenosis is slow and weak and it does not change in whatsoever position the arm is placed. In mitral stenosis the pulse is weak and if the arm is held in a vertical position over the head the pulse may disappear. In mitral regurgitation the pulse is weak, when the arm is held in a vertical position and reappears strongly when held in a horizontal position. When compensation is lost the pulse in mitral disease disappears entirely upon raising the arm over the head.

In radial arteriosclerosis these characteristics may not appear, and the radial pulse may be so weak in any position as to be hardly appreciable. Errors may occur if two murmurs are heard at the same time. The only diastolic murmur occurring in the aged is the aortic regurgitant murmur. The aortic obstructive and the mitral and tricuspid regurgitant murmurs occur during the systole, but the aortic murmur is never heard at the apex, the mitral murmur is heard over the apex and to the left and is often heard at the back, the tricuspid is heard to the right of the sternum. As diastole begins with the beginning of the second sound we must be certain about the two sounds and if there is any doubt as to which is the first and which the second we must find the apex beat or the carotid pulse, thus determining the systole. The radial pulse is useless for this purpose. An hour-glass murmur, one which is heard loudest at one valve, gradually growing weaker to a certain point, then again increasing in intensity as we approach another valve, indicates that the two valves are diseased. In the aged this happens most frequently in aortic obstruction and mitral regurgitation.

Presystolic murmurs are heard best when the patient is sitting or standing up, systolic murmurs are heard best when the patient is lying down and the murmur of tricuspid regurgitation can sometimes be heard only in Stern's posture, lying down with the head slightly lowered so as to stretch the vessels of the neck. Diastolic murmurs are not affected by position.

All murmurs heard in the region of the heart are not due to

valvular lesions nor do all cardiac murmurs imply diseased valves. There may be a relative insufficiency in which the orifice is enlarged so that the valve cannot close it completely, yet the valve will be sound. The Austin Flint murmur sometimes heard near the apex in aortic regurgitation may simulate the murmur of mitral stenosis and is in the locality in which mitral murmurs are usually heard, yet it has apparently no connection with mitral disease.

Functional or accidental murmurs are infrequent in the aged, as the probable cause, pressure upon or suction of the overlapping edges of the lungs by the cardiac contractions, does not prevail where the lungs are atrophied. In anemia or debility, leakage may occur through the mitral or tricuspid valve. This real, though temporary relative insufficiency is not an organic defect. The pericardial friction sounds and cardiorespiratory murmurs are affected by the respiration and there is a history pointing to the underlying cause.

AORTIC REGURGITATION

The author has found this one of the most frequent valvular lesions in the aged, while other observers consider mitral regurgitation to be the most prevalent one. Aortic regurgitation is in some cases a relative insufficiency caused by dilatation of the orifice, more often, however, the fault lies in the valve itself.

Etiology.—Relative insufficiency is due either to a dilatation of the aorta or aortic aneurysm just above the valve, thereby stretching the aortic ring, or to dilatation of the left ventricle. In insufficiency due to valvular defect the cause is either the general cause of ageing and arteriosclerosis affecting primarily the valve, or it may arise from the extension of an arteriosclerosis of the aorta or from senile endocarditis.

Pathology.—In relative insufficiency the valve is not affected. The orifice is distended, and when the valve closes, the edges do not approximate. When the fault lies in the valve the cusps thicken, harden, contract and shrivel up, or they may adhere to the endocardium. The left ventricle receives normally from the auricle the quota which will completely fill it. When more blood enters the heart after rebounding from the aorta the muscle fibers become stretched and the cavity dilates.

As long as the hypertrophy is sufficient to compensate for the disturbed circulation, no further change occurs. When the limit of functional ability is reached and decompensation begins, dilatation proceeds rapidly, an insufficient supply of blood reaches the coronaries and myocardial degeneration sets in through impaired nutrition. The cordæ tendinæ shrink and cause relative insufficiency of the mitral valve while the valve itself is weakened through the extra strain placed upon it by the greater amount of blood in the ventricle. It also degenerates primarily from senile involution or secondarily from extension of senile endocarditis. This causes pulmonary and venous engorgement with the consequent train of symptoms described in cardiac dilatation.

Symptoms.—There may be no subjective symptoms while compensation is complete, though objective symptoms and physical signs are manifest. A pathognomonic symptom is the pulse, called Corrigan's or water hammer pulse. The increased force required to send an excessive amount of blood from the heart produces a strong, full pulse, but as some of the blood returns to the ventricle the pulse strength drops rapidly and fades away. This characteristic can be brought out very early in the disease, if the arm is lifted above the head, allowing the force of gravity to aid in the rapid emptying and collapse of the radial artery. Other early symptoms are increase in carotid pulsation and the usual symptoms of cardiac hypertrophy. When decomposition sets in there are the symptoms of cardiac dilatation, with dyspnea, precordial distress, and the symptoms of venous engorgement.

The earliest physical sign is usually a visible carotid pulsation. The heaving impulse given to the chest is not as marked in aged individuals as in earlier life owing to the rigidity of the chest wall, but there may be a pronounced bulging. The apex beat may be weaker and more diffused. Capillary pulsation is usually an early sign and may be demonstrated by rubbing the thumb nail across the forehead, when the hyperemic line will redden and then become paler with each pulsation. Percussion gives little information of value, as the heart is usually hypertrophied in the aged, but in advanced cases the hypertrophy and consequent dilatation are greater than in any other cardiac disease.

A diastolic murmur in the aged is virtually pathognomonic of aortic regurgitation. Other diastolic murmurs are rare in old people and can readily be distinguished from the murmur of aortic insufficiency. Pulmonary regurgitation which gives a diastolic murmur may set in when decomposition is complete and all the valves are involved. By this time the many confusing abnormal sounds heard in the chest, the diversity in the areas and direction of transmission, the arrhythmia and signs of profound pulmonary and circulatory disturbances, make a definite differential diagnosis impossible. Other diastolic murmurs which might possibly occur in the aged are a cardio-respiratory murmur and a venous hum, due to anemia. The former disappears when holding the breath, the latter disappears when slight pressure is made upon the jugulars. (Prolonged pressure may induce cerebral hyperemia and hemorrhage.) A diastolic pericardial friction sound may be heard at the base of the heart in acute adhesive pericarditis, but it is incidental to the pericarditis, the sound is usually double and the symptoms of aortic disease are absent.

Mitral stenosis which gives a presystolic murmur may be mistaken for aortic regurgitation. The former immediately precedes the first sound, the latter immediately follows the second sound. Mitral stenosis is rare in the aged except as a secondary and late sequel to aortic disease, mitral regurgitation or general arteriosclerosis.

Numerous other symptoms and signs may occur in aortic regurgitation, but they add nothing to the determination of the diagnosis of this disease in the aged. The so-called pistol-shot sound, a systolic sound heard over the arteries, especially over the femoral, and caused by the sudden filling of the empty vessel, is rarely appreciable. Duroziez' sign, a diastolic murmur heard over the larger vessels when pressure is made by the stethoscope is rarely heard. Traube's sign, a double murmur heard over the carotid and femoral, is rare and indistinct. The Flint murmur, a faint rumble heard at the apex immediately after the murmur of the aortic regurgitation, is occasionally met with, but if occurring as a presystolic murmur it can hardly be distinguished from the murmur of mitral stenosis except by the absence of signs of pulmonary engorgement. The prognosis is good as long as the hypertrophy maintains full compensation.

In most cases the excessive work imposed upon the heart in sending the blood through sclerosed vessels brings it sooner or later to the limit of its functional ability, while the internal pressure in the left ventricle produced by an excess of blood causes dilatation. The mitral valve becomes involved and the disturbance in the pulmonary and circulatory systems hasten degeneration of other organs. While the mitral insufficiency is a temporary safety valve for the dilated ventricle by relieving the overdistention, it causes pulmonary engorgement.

Dilatation of the aorta is generally associated with aortic regurgitation, and mitral regurgitation is found in almost every advanced case. This is generally followed by mitral stenosis and aortic stenosis, later by tricuspid regurgitation and other valvular defects. The sequence may, however, be altered in myocardial degeneration.

Treatment.—See Treatment of Cardiac Lesions.

AORTIC STENOSIS

Aortic stenosis, though occurring quite frequently in old age, is always a complication of some other valvular lesion, generally aortic regurgitation. Cabot reports that out of over 250 autopsies in cases of valvular disease there was not a single uncomplicated aortic stenosis but there were 29 occurring with aortic regurgitation.

Etiology.—This disease in advanced age is due to extension of atheroma from the aorta, extension of senile endocarditis, or atheromatous deposits about the aortic ring, or it may occur as a degeneration originating in the valve itself.

Pathology.—The cusps become thick and rigid and sometimes calcareous plates form under the valve, which becomes misshapen and obstructs the free passage of the blood out of the ventricle, while the cusps, not closing completely, allow blood to return from the aorta, thus causing an insufficiency. This is the usual condition of the aortic valve when diseased. In some cases the margins of the cusps adhere to each other, thus reducing the opening. Occasionally there are fibrin deposits upon the valves which prevent the complete opening of the cusps, or which present rough edges or free ends to the stream. Hypertrophy is invariably present and late in the disease there is dilatation.

Symptoms.—The pathognomonic symptom of aortic stenosis is the pulse, "*pulsus rarus, parvus tardus,*" infrequent, small and slow, in contrast to a strong apex beat. There is a systolic murmur, heard best in the third intercostal space close to the right margin of the sternum, the sound being carried upward toward the neck. If a murmur is heard at the apex also it is due to another valvular lesion. The area of cardiac impulse is increased, but the apex impulse is indistinct, the force of the heart is increased also, and a purring thrill is felt over the heart, especially marked over the valve, and sometimes felt in the carotids, and there is an increased area of dulness to the left and downward showing hypertrophy of the left ventricle.

Other conditions, which give a similar thrill and murmur, are aortic aneurysm and aneurysm of the innominata, and these can readily be differentiated by the absence of the diastolic shock, tumor, abnormal pulsation, pain, symptoms of pressure upon the trachea, bronchi or recurrent laryngeal nerve, etc., found in aneurysm, and by the presence of the characteristic pulse. A diffuse dilatation of the aorta may give a systolic murmur, but this condition is almost always associated with aortic regurgitation and an increased area of dulness above the base. Roughening of the aortic valve may give a systolic murmur over the valve. This is distinguished from the murmur of stenosis by the accentuation of the second sound of the heart, while in stenosis this sound is faint. If the intima of the aorta is roughened there may be a systolic murmur at the base, but there is no thrill and the characteristic pulse is absent. Functional systolic murmurs are rare in the aged and they do not present any of the other signs of aortic disease. Pulmonary stenosis occurs only when all the valves are involved in decompensation. The distinguishing signs between aortic and pulmonary stenosis are useless in the aged, as the latter never occurs without the aortic and other cardiac lesions.

Mitral regurgitation has a systolic murmur which is heard most distinctly over the apex, but there is no thrill nor the characteristic weak slow pulse. Tricuspid regurgitation has a systolic murmur but none of the other signs of aortic stenosis and there is generally a jugular pulsation and pulmonary disturbance. Aortic stenosis is almost invariably associated with

aortic insufficiency and the prognosis depends upon the prognosis of the latter disease.

For **Treatment** see Cardiac Lesions.

MITRAL REGURGITATION

Mitral regurgitation is generally either a degenerative condition, or it is a relative insufficiency secondary to dilatation. In many cases both conditions prevail.

Etiology.—Relative insufficiency occurs when myocarditis or dilatation stretches the orifice so that the flaps cannot approximate fully. This generally occurs after aortic regurgitation and this combination of valvular lesions, aortic and mitral regurgitation, is the first result of decompensation following the primary lesion. In rare cases the senile degenerative process begins in the valve, more often it follows an extension of senile endocarditis or shortening of the cordæ tendinæ. The etiological factors which make it the most prevalent lesion in earlier life, rheumatism and excessive physical activity, rarely appear in later life.

Pathology.—The valve becomes thickened, hardened and shortened. The cordæ tendinæ thicken and shorten and drag down the flaps. The papillary muscles grow thinner. The valve flaps may adhere to the ventricular wall and thus become immobilized. The excess of blood in the auricular chamber causes the walls of that chamber to dilate. The walls at the same time hypertrophy and for a time compensation is maintained, but the dilatation proceeds faster than the hypertrophy and the latter soon reaches the limit of its capacity. After decompensation sets in, the blood is dammed back into the lungs and later the right side of the heart becomes affected. There is first hypertrophy, later, dilatation of the right ventricle, tricuspid regurgitation, right auricular hypertrophy, then dilatation, obstruction to the venous circulation, with degeneration following passive congestion of the liver, kidneys, stomach, spleen, etc. Late in the disease, hypertrophy, then dilatation of the left ventricle occurs, and every valve and chamber of the heart is affected.

Symptoms.—There are no early symptoms or signs of mitral regurgitation except a systolic murmur at the apex, the sound

being carried to the back behind or below the angle of the scapula. The apex beat is found more to the left than normal and the area of dulness is increased downward and to the left. The second sound of the heart is more intense over the pulmonic valve. When dilatation and decompensation ensues, there follows the train of symptoms described under cardiac dilatation. The earliest of these symptoms is cardiac asthma, dyspnea, palpitation and slight cyanosis upon exertion, and sometimes even without exertion. Later the dyspnea and cyanosis are constant and venous engorgement of the liver, kidneys, brain and stomach follow. The disturbance to the pulmonary circulation produces dyspnea, cyanosis and a cough with watery and occasionally blood-stained expectoration. The liver becomes enlarged, there is a feeling of weight and pain in the right hypochondrium and there may be jaundice. Kidney involvement is shown in scanty high-colored urine, sometimes albuminous and occasionally presenting casts. Cerebral hyperemia is produced with headache, vertigo, occasional stupor or even delirium. There is gastric and intestinal catarrh and usually hemorrhoids. Later, as a result of the circulatory disturbance, dropsy and anasarca set in. The pulse becomes weak and, upon excitement, it is irregular in rhythm and force.

Diagnosis.—The only other systolic murmurs that may be confused with that of mitral regurgitation are functional murmurs and the murmur of tricuspid regurgitation. Functional murmurs are rare in the aged, they are faint or absent while in the upright position and are transmitted to the back. The murmurs of aortic stenosis and roughening of the aortic valve are heard best over the valve and are not transmitted to the back. Aortic stenosis has a characteristic pulse which is absent in the mitral disease.

Tricuspid regurgitation gives a murmur so similar to that of mitral regurgitation that it is often impossible to differentiate between them and as the tricuspid lesion always occurs after the mitral lesion it is generally overlooked. Tricuspid murmurs are not heard in the back and their maximum intensity is to the right of the sternum, but they may be heard distinctly at the apex. These points are, however, useless for the determination of the diagnosis, as the mitral lesion is invariably present. Jugular pulsation, an early symptom of

tricuspid regurgitation, does not occur in uncomplicated mitral regurgitation. If it does appear in mitral disease it is an evidence of tricuspid involvement.

Treatment is given under Cardiac Lesions.

MITRAL STENOSIS

Mitral stenosis is a degenerative process in which the shrunken and thickened flaps diminish the size of the auriculo-ventricular opening.

Etiology.—The causes prevailing in earlier life do not prevail in old age and when the disease occurs in earlier life the individual does not live long enough to grow old. Mitral stenosis, when occurring in the aged, is almost always due to a degenerative process originating in the valve itself or carried to the flaps by extension from senile endocarditis. It may follow Bright's disease and Vinay reported a case in which a deposit of calcareous matter about the ring caused a diminution in the size of the orifice.

Pathology.—The flaps become thickened, hardened and shrunken. The shrinking of the valves prevents complete apposition of the edges with consequent mitral insufficiency. The button-hole slit caused by adhesion of the edges of the valve is rarely found in advanced life. This adhesion may be due either to a change in the blood which permits fibrin to be deposited upon the edges with consequent agglutination of these deposits, or else to an irritation with following adhesive inflammation of the edges, from blood toxins. The latter cause would also explain the presence of endocarditis wherever we find the button-hole slit. The only disease causing such irritation in old age is chronic nephritis and it is only when this disease is present that the button-hole slit is found. When the stenosis progresses and mitral regurgitation ensues, there follow hypertrophy and dilatation of the left auricle, with blocking of the pulmonary circulation and with the train of disorders that follow pulmonary engorgement.

Symptoms.—Mitral stenosis is a lesion that may exist for years without giving any marked symptoms or signs. Among the earliest of the suggestive symptoms is irregularity of the heart in rhythm and force upon exertion which rapidly subsides

after resting. Occasionally a purring thrill may be felt at the apex just preceding the apex beat. As the disease is almost invariably associated with mitral insufficiency, the symptoms of stenosis may be completely masked by those of the insufficiency and as soon as decompensation sets in there are the usual symptoms of pulmonary, and later, venous engorgement. The only symptom pointing to mitral stenosis is frequent hemoptysis. The murmur of mitral stenosis is almost pathognomonic. It is a presystolic murmur, loud, long and rumbling, ending with the first sound of the heart, and heard in a limited area about the apex. The only other murmur which may possibly be mistaken for it is the Flint murmur, which occurs occasionally in aortic regurgitation and tricuspid stenosis, a very rare condition, unknown in the aged.

The murmur of mitral stenosis is frequently absent in the early stage of the disease or it may appear temporarily during or immediately following exertion. It also disappears after decompensation sets in. The purring thrill about the apex can sometimes be felt before the murmur appears and it, too, disappears as soon as decompensation occurs. It is presystolic, and ends with the apex beat.

The first sound of the heart is short, loud and snapping; the pulmonic second sound is accentuated and is sometimes double. When decompensation occurs or when other valvular lesions exist, the short, sharp first sound may be the only sign determining the presence of a mitral stenosis.

The prognosis of mitral stenosis in the aged depends upon the mitral regurgitation present. The two may exist for years without giving distress, but sooner or later decompensation sets in and carries the patient rapidly to the fatal issue.

Treatment.—See Treatment of Cardiac Lesions.

TRICUSPID REGURGITATION

Tricuspid insufficiency, though rarely diagnosed, is a frequent sequel to mitral disease in the aged. In these cases it is a relative insufficiency, caused by a dilatation of the right ventricle with stretching of the auriculo-ventricular orifice. A degeneration of the valve is possible, but out of over 400 autopsies made in Guy's Hospital in which tricuspid insufficiency

was found, there was not one showing valvular degeneration. In some cases it was due to myocarditis or to pulmonary disease.

Symptoms.—The symptoms are referable to venous engorgement. Specially marked in this disease are systolic jugular pulsation or vibration, distention of the superficial veins of the upper part of the body when the patient coughs or strains, and cyanosis which is more pronounced when the patient is in the horizontal position. When in this position cerebral hyperemia occurs and may be of such extent as to produce cerebral compression with stupor and coma. Symptoms associated with passive congestion in other organs appear, especially in the liver and kidneys. A pulsation over the liver is pathognomonic of tricuspid regurgitation, but it is often absent. A jugular pulsation or vibration may be normal or due to other causes. If a vein, after being temporarily emptied by stroking from below upward, immediately fills up from below, it is pathognomonic of tricuspid regurgitation. If it does not fill from below, the cause of the pulsation is not due to this disease. Tricuspid regurgitation presents definite physical signs but in the aged this condition is almost invariably associated with mitral regurgitation and the more marked signs of the mitral disease may mask the signs of the tricuspid lesion. There is a systolic murmur near the fifth left costal cartilage, not loud, and often absent. In some cases it may be brought out in Stern's position, *i.e.*, the patient lying horizontally with the head slightly lowered. The area of dulness is increased to the right of the sternum.

The **diagnosis,** in the absence of the murmur, depends upon the pulmonary symptoms, dyspnea and cyanosis which become worse in a horizontal position. Jugular and epigastric pulsation confirm the diagnosis.

Treatment.—See Treatment of Cardiac Lesions.

COMBINED VALVULAR LESIONS

It frequently happens that two or more valvular lesions exist at the same time. One valvular lesion may cause a defect in another, or the same valve may produce both stenosis and regurgitation. Such combined valvular lesions produce a variety of symptoms and signs, some of which may modify others.

The most frequent of the combined lesions is aortic regurgitation and a relative mitral insufficiency produced by the ventricular dilatation following the aortic defect. Tricuspid relative insufficiency follows a mitral regurgitation. If there is a mitral stenosis, a mitral regurgitation accompanies it. Aortic stenosis is generally associated with aortic regurgitation or is followed by mitral regurgitation. When compensation is lost there may be aortic, mitral and tricuspid lesions with murmurs all over the chest, and arrhythmia, irregular pulse and irregular cardiac impulse, making it impossible to determine what valves are affected or how.

In *aortic* and *mitral regurgitation* a murmur is heard after each sound, the murmur after the first sound being most distinct at the apex, the diastolic murmur being loudest at the third costo-sternal articulation. The murmurs are transmitted downward and to the left. The water hammer pulse is modified; it is weaker and does not fade away as rapidly as in the uncomplicated aortic regurgitation. Capillary pulse may be absent. In *aortic stenosis* and *regurgitation* there is a double murmur over the aortic area, one after the first sound, the other one following the second sound of the heart, the first being transmitted upward toward the neck, the other downward toward the xiphoid cartilage. The pulse is modified as in mitral complication, capillary pulsation is diminished and the pulsation in the peripheral vessels may be absent. The thrill of aortic stenosis may be absent also.

In *double mitral disease* the murmur precedes and follows the first sound of the heart. The presystolic murmur is often absent and must be brought out by some exertion such as a fast walk or jump. If once heard the diagnosis is certain and as mitral obstruction is always associated with regurgitation, the symptoms of the latter disease need not be sought for.

Mitral obstruction does not occur with aortic disease. *Aortic stenosis* may be associated with *mitral regurgitation*. There is a systolic hour-glass murmur in this case, the one maximal point being at the apex, the other at the second right costo-sternal articulation or just below it with a vanishing point at the fourth left costo-sternal articulation. The pulse of aortic stenosis is not altered, but the thrill is weakened.

Mitral regurgitation and *tricuspid regurgitation* are often

found together but it is necessary to determine the presence of the tricuspid lesion alone. The murmurs of the two occur at the same time their areas of conductivity overlap and their points of maximum intensity are so close together that errors are very liable to occur. The diagnosis must be made by the signs of mitral regurgitation and by the symptoms of the tricuspid disease, like jugular pulsation, etc.

TREATMENT OF CARDIAC LESIONS

In the treatment of cardiac lesions in old age this rule is imperative: *No interference while compensation is complete.* Proper hygiene and the avoidance of sudden or prolonged strain must be observed to prevent rapid loss of muscle tone, and there should be no medicinal treatment as long as the heart maintains its strengh and regularity.

The aged person should continue to take exercise and may continue at his ordinary vocation if it does not involve sudden strains, but the moment dyspnea or palpitation appears he must stop and lie down. He must guard against straining at stool and should avoid foods which tend to produce flatulency. Intense mental concentration and powerful emotions, even if pleasurable, are injurious, while shock may cause sudden death.

Failing compensation comes on gradually and its earliest symptom is slight dyspnea without previous exertion. As soon as this is noticed the patient should go to bed and remain there for at least a week. Compensation is occasionally restored by prolonged absolute rest. In administering drugs in cardiac disease of the aged, there are a few general rules that must be observed. Heart tonics should not be given until they are required to overcome weakness, and heart stimulants should never be given except in an emergency or to counteract rapidly acting cardiac depressants. Vasoconstrictors must be used cautiously, as the sclerosed vessels may not be able to withstand increased pressure. Heart depressants are of service only in hypertrophy without valvular lesion. Drugs are rarely required in this condition unless the hypertrophy is extensive enough to cause palpitation and active cerebral congestion. In this case we can use aconite in 1-minim doses several times a day until the heart action is lowered. Veratrum viride or gel-

seminum in 5-minim doses of the tincture can be given in the place of aconite. (Chloral hydrate should not be used in old age.) In some cases of aortic stenosis aconite or veratrum will steady the heart but the drug must be stopped as soon as an effect is produced. Digitalis, the most valuable drug in the pharmacopeia, is dangerous in senile conditions. It is contra-indicated in aortic disease, myocardial degeneration and in cases of high blood pressure. When given by mouth the response in a stronger pulse appears about twelve to thirty-six hours later; therefore, it is useless when quick action is desired. It must be noted that its action is cumulative. The author has seen two cases of apoplexy following the hypodermic use of digitalis preparations in threatened heart failure. If the heart is rapid and weak tincture of strophanthus should be given in 2- to 4-minim doses. Adonin in 1/8-grain doses several times daily can be used in aortic regurgitation. Tincture of cactus grandiflorus in 5-minim doses and of convallaria 5- to 10-minim doses can be given in place of digitalis and strophanthus. The frequent lack of success when using these drugs must be ascribed to their poor quality. More positive results are obtained when using their glucosides, convallamarin and cactin. The special indication for spartein is a slow, weak heart, such as occurs in decompensation following aortic stenosis. This should be used in 1/2- to 2-grain doses until the heart beats respond with greater strength and frequency, when it must be discontinued. It may be combined with 1/50-grain strychnia. The nitrites, amyl nitrite, spirit glonoin and sodium nitrite are powerful vasodilators and should be used only to relieve cardiac spasm, an overdistended heart or poor peripheral circulation. They should not be used if the face is flushed or where there is cerebral hyperemia. The amyl nitrite used in 3- to 5-minim doses by inhalation is of service in angina pectoris, cardiac asthma and syncope. In threatened heart failure nitroglycerin in 1/100-grain dose can be used hypodermically combined with 1/30-grain strychnine and 1/100 grain digitalin. For prolonged action the nitrite of soda can be given in 1/6- to 1-grain doses every four hours.

Other emergency drugs are to be given only where there is danger of cardiac exhaustion; these are carbonate of ammonia in 5-grain doses, compound spirits of ether 30 minims, or sul-

phuric ether hypodermically in 20-minim doses, camphor 5 grains in oily solution hypodermically, and alcohol. The most rapid action is obtained from the hypodermic use of ether but the action is evanescent. Strychnine should not be used before decompensation sets in, as it simply hastens decompensation and it should be used only as an emergency drug, being a cardiac stimulant and not a cardiac tonic. The distressing symptoms accompanying cardiac lesions require treatment distinct from that of the disease.

For dyspnea, if there is no contraindication to the nitrites, the nitrite of amyl gives most prompt relief. Morphine or dionin in 1/8-grain doses is of service for dyspnea occurring at night and preventing sleep. However, neither the nitrites nor morphine should be used continuously. For prolonged treatment the tincture of cimicifuga in 1-dram doses, combined with arsenic in the form of Fowler's solution in doses of 3-minims, should be used three times a day. The arsenic must be discontinued when its physiological symptoms appear.

Cardiac asthma will generally yield to 1/2-dram doses of compound spirits of ether. Palpitation or arrhythmia will sometimes subside upon a hypodermic injection of 1/120 grain of atropia. If it occurs frequently the bromides may be given. The selection of the drug must depend upon the condition of the heart, using sedatives in hypertrophy, the nitrites if there is high blood pressure, and morphine, spartein, strychnine, ice-bags, etc., as indicated. In some cases when decompensation sets in suddenly with dyspnea and palpitation, immediate rest in bed, remaining there for several days while eating and drinking sparingly, will restore compensation.

In insomnia a hot foot bath should be tried. If this fails we can use 5 to 10 grains of veronal and if there is considerable mental agitation it should be combined with 3 grains of monobromated camphor. *No Chloral!* Morphia may be used if veronal fails, but it must be combined with atropin to prevent paralysis of the respiratory centers.

Nothing will relieve the cyanosis except the inhalation of oxygen and this is of service only while it is being used. It should be employed occasionally in cyanotic cases to improve the condition of the blood, and to give temporary relief to the lungs and thus indirectly to the brain. Edema, which occurs

late, unless associated with nephritis, is difficult to treat in the aged. A salt-free diet is the most important measure. For this purpose malted milk with milk or water in small quantities, just sufficient to supply the actual needs of the system, should be taken. Mild diuretics should be used. In cardiac dropsy calomel in 1/10-grain doses every three hours and 4 drams potassium bitartrate every second day will produce free diuresis and catharsis. The legs should be elevated and when the edema disappears they should be bandaged, but the bandages must not be too tight, as they might compress sclerosed vessels and obliterate them. Puncturing the edematous limbs is useless in the dropsy due to tricuspid regurgitation as edema reappears in a few days, but it may become necessary to puncture an abdominal dropsy. If chronic nephritis complicates the cardiac disease diuretics should be used cautiously, lest they increase the irritation to the kidney. It is better in these cases to try to reduce the edema by a salt-free diet and hydragogue cathartics, though the latter further impair the already impoverished blood.

The Oertel, Schott, and Nauheim treatments of heart disease are contraindicated in old persons.

INTESTINAL OBSTRUCTION

Partial obstruction is generally overlooked. It may be temporary or permanent, coming on slowly, and gradually increasing until complete occlusion occurs, or it may come on rapidly and, if due to impaction, is quickly relieved by removal of the offending substance. Complete occlusion comes on suddenly and unless soon relieved is rapidly fatal. The two conditions, stenosis and occlusion, will be described separately.

Intestinal Stenosis

Etiology.—The most frequent cause of temporary stenosis in later life is impaction with feces or gall-stones, while a permanent stenosis is usually due to enteroptosis. Other causes are partial occlusion by growths within the bowel, or pressure from adjacent tissues upon the bowel; thickening due to inflammation; contraction following such inflammation; contrac-

tion or growth of scar tissue over the site of an ulcer, or other lesion; kinks, volvulus, hernial constrictions, strangulation by bands, adhesions to the peritoneum or mesentery, etc. Intussusception is rare in the aged. Many of these causes may produce complete occlusion. Obstruction of the bowel may also be due to primary intestinal paresis or it may be due to a hemiplegia or paraplegia, the paralysis producing a paralysis of the intestines with consequent intestinal impaction.

Symptoms.—The symptoms of intestinal obstruction depend upon the cause, degree and location of the obstruction. Complete obstruction will be considered in the chapter on Intestinal Occlusion.

The location of the obstruction can often be determined by inspection and palpation, occasionally by other signs. If the duodenum or jejunum is blocked, the upper part of the abdomen is distended, digestive disorders occur early, vomiting may occur but it has no fecal odor. The urine is diminished or may be entirely suppressed. If obstructed in the region of the ileocecal valve the distention is greatest about the umbilicus and there is early fecal vomiting, or, if the obstruction is not severe or complete, there may be eructations of gas having a fecal odor. Peristaltic movements are often observable over the upper part of the abdomen. If the obstruction is in the large intestine the distention is at the sides and lower part of the abdomen. Obstruction due to impaction of feces or other substances, or to growths, can generally be diagnosed by the presence of a mass at the point of obstruction. A doughy mass which pits upon pressure and remains pitted is feces; a hard mass may be gall-stones or enteroliths; a soft mass which does not pit is a growth. There may be impaction above a growth and the palpation signs of both will manifest themselves. An intestinal growth is movable with the bowel. If outside of the intestines it can be separated from the bowel. Complete occlusion of the large intestine can be determined by testing the capacity of that portion of the gut. The rectum holds normally three pints, the colon holds six quarts of water. If the entire amount can be introduced the obstruction is above the colon.

Enteroptosis is frequently found in the aged in connection with ptoses of other abdominal viscera. It is due to weakened

mesenteric attachment, flaccid abdominal walls, the weight of other displaced viscera, and possibly, to the weight of feces in the transverse colon. There are no clearly defined symptoms, and a positive diagnosis can be made only by radiography. The presence of a gastroptosis which can often be diagnosed by percussion and sometimes by inspection, associated with a sense of weight in the pelvis and with protrusion of the lower part of the abdomen, point to enteroptosis. There is also usually backache, flatulence and frequent urination. It is sometimes possible to inflate the colon and its location can then be made out providing the abdominal walls are thin. The symptoms pointing to partial obstruction in the displaced gut are constipation, a powerful effort being required to force the stool out, occasional watery diarrhea containing scales of hardened feces which irritate the sphincter ani and distention of the bowels with gas and gurgling sounds in the lower part of the abdomen. There are often colicky pains, especially when making an effort to propel the feces, which are passed in a ribbon or pencil form, depending upon the shape of the stenosed aperture. Impacted feces can sometimes be felt in the right iliac fossa.

Impaction of feces generally occurs in the descending colon. The mass can be felt and distinguished by its doughy consistency, the impress made upon the mass by the pressure of the finger remaining after the pressure is removed. There are colicky pains and tenesmus, abdominal distention by gas, constipation, stools occasionally passing in small scales and irregular lumps. In some cases feces will force a channel through the impacted mass and will then come away in small cylinders or balls. It is thus possible to have a daily stool with fecal impaction. Fecal impaction will give the constitutional symptoms of autointoxication due to the reabsorption of fecal matter. *Impaction* by *gall-stones*, *enteroliths* and other foreign bodies generally occurs either at the ileocecal valve or in the upper part of the bowel. These may sometimes be felt as hard masses, not doughy, if the abdominal walls are thin and the intestines are not distended with gas. They produce considerable colic and digestive disturbance but, unless they completely occlude the gut, they allow the passage of semiliquid fecal matter, which reaching the lower bowel becomes formed and passes as a normal stool. If due to gall-stones there will

be the symptoms pointing to them and bile will be found in the stools. Enteroliths and other foreign bodies give no distinctive signs by which their nature can be determined. The presence of a mass which cannot be pressed into or moved without dragging along adjoining tissues, eliciting a dull ache, but no sharp pain upon pressure, and giving the other symptoms of intestinal obstruction points to a foreign body partly occluding the intestines. The X-ray gives the most definite information. *Growths* may occlude the lumen of the bowel entirely or partly. Benign growths increase slowly, the symptoms are mild and may pass unrecognized until complete occlusion occurs. The intestines may accustom themselves to the increased pressure at the point of constriction and a compensatory dilatation opposite to the growth may result. In such cases the feces will appear ribbonlike. Malignant growths occur most frequently in the colon and rectum. They are readily diagnosed by their rapid progress, pain, cachexia, emaciation, involvement of other tissues, etc. *Cicatricial stricture*, and *contractions, following inflammation* cannot be specially diagnosed. They may be suspected where there is a pencil-shaped stool with the usual symptoms of partial obstruction, constipation with occasional stools, tenesmus, distention of the bowels, and colicky pains; these symptoms slowly increasing in severity. *Adhesions* to the peritoneum or mesentery may occur and give rise to partial obstruction of the gut with the ordinary symptoms of that condition. There are no pathognomonic signs and the diagnosis must be made by the history of a former peritonitis and exclusion of other causes. The symptoms are usually mild, in some cases consisting only of an irregular constipation and occasionally a feeling of dragging upon the abdominal walls. *Paresis of the intestines*, if occurring suddenly, gives the symptoms of complete occlusion. A slow progressive paresis begins with constipation, a gradually increasing difficulty in emptying the bowels and finally complete inability to move them followed by all the symptoms of complete occlusion. Kinks described by Lane may occur in any part of the intestines and produce a partial obstruction with chronic intestinal stasis. This does not give the usual symptoms of obstruction, but the symptoms of a slow persistent antointoxication. It rarely proceeds to occlusion. Strangulation by bands, or hernial con-

tractions, volvulus and intussusception almost always cause complete occlusion at once or within a few hours.

Prognosis.—The prognosis of intestinal stenosis depends upon the cause. Fecal impaction can generally be removed and the obstruction disappears. Enteroptosis and adhesions to the peritoneum are permanent. They rarely produce much distress, rarely progress to complete occlusion and do not endanger life unless complete occlusion occurs. Benign growths and cicatricial stricture proceed very slowly to complete occlusion, while malignant growths rapidly close the gut, and such growths outside of the intestine, but in close juxtaposition to it, will frequently involve the bowel. Obstruction due to other causes generally proceeds rapidly to complete occlusion.

Treatment.—Medicinal measures are of service only in impaction of feces or gall-stones and for the temporary relief of distressing symptoms. If the fecal impaction exists in the colon, it may be softened by introducing a steady stream of warm water to which bicarbonate of soda, salt and glycerine has been added in the proportion of an ounce of each to the quart. The syringe should not be held high but the flow should be steadily maintained. The first part of feces passing away immediately after the enema are but the contents of the rectum and not the impacted mass. It is sometimes possible to reach the impaction with a rectal scoop but unless the whole mass can be scooped away the enema should be repeated until softening of the impacted feces has been accomplished. Massage is dangerous as injury to the intestinal wall can be easily produced by rough handling. An enema will not go beyond the colon and impaction beyond that must be reached by internal medication. The fecal mass above the ileocecal valve being fluid, impaction beyond that must consist of gall-stones or a foreign body. The following mixture has served well in such cases. Croton oil, 1 minim, castor oil 1/2 ounce and olive oil 3 1/2 ounces, taken in one dose to be repeated in four hours if necessary. A minim of oil of peppermint and half a grain of saccharin will disguise the smell and taste. If there is a rapid intestinal paresis, eserine in 1/50-grain doses may be used hypodermically and repeated in three hours. Slow paresis must be overcome by the internal administration of persistaltic stimulants.

In those cases in which there is a permanent or slowly advancing partial occlusion, the most important measure is the regulation of the diet. The food should be easily digestible and leave little waste. Food containing much cellulose, seeds, rind, nuts and all other substances that cannot be completely converted must be avoided. If there has been a persistent constipation for several days, the predigested or readily converted foods should be used, but those containing a large percentage of alcohol must not be taken. Malted milk is perhaps the best for prolonged use, but if a change is desired any of dry and non-alcoholic liquid foods can be used. If a cathartic is required castor oil will act best. When complete occlusion occurs surgical intervention is necessary.

Intestinal Occlusion

Etiology.—Any cause that can produce partial intestinal obstruction may also produce complete occlusion, and many cases of partial obstruction proceed to complete occlusion. The most frequent causes of intestinal occlusion in the aged are cancerous growths and hernia. Volvulus and kinks are also frequent primary causes. Secondary causes are paralysis of the bowel associated with hemiplegia or paraplegia or following traumatism; peritoneal bands following peritonitis; scar tissue, completely closing the gut; occasionally impaction, rarely intussusception.

Symptoms.—The symptoms of intestinal occlusion come on suddenly, with severe abdominal pain, which is at first colicky, but later becomes continuous. There is generally a history of constipation and in some cases the history will show a progressive partial obstruction which had reached the stage of complete occlusion. Shortly after the pain sets in, vomiting begins without nausea or straining. At first the contents of the stomach are ejected and afterwards a greenish or brownish bile-stained fluid. If the occlusion occurs in the duodenum the ejected matter continues to be bile-stained but is not feculent. The fecal odor becomes more marked the farther along the gut the obstruction occurs. Tympanites begins about the second day and becomes extreme and feculent vomiting sets in about the third day. Collapse may occur as soon as the

initial pain is felt. Usually, however, there is rapidly deepening mental and physical depression, while collapse sets in only after the stercoraceous vomitus appears.

In some cases, after the initial pain is felt, there is a watery diarrhea lasting a few hours then complete constipation. Tenesmus is severe if the occlusion is in the large intestines and repeated efforts at stool may being forth a little blood-stained mucus. The urine is either suppressed or it is scanty and contains indican and phenol. The mind is clear but there is an intense depression associated with the fear of dying. The Facies Hippocrates may appear within a few hours after the occlusion has occurred. A tumor can sometimes be felt at the site of the occlusion, while above that point the intestine bulges out so that it can be seen and felt.

A *cancerous growth* is usually recognized long before it has produced complete occlusion of the gut. It is generally located in the colon or rectum; but occasionally a cancer of the mesentery will produce intestinal occlusion. *Hernia* may be either intraabdominal, or extraabdominal, umbilical, inguinal or femoral. It is impossible to diagnose an intraabdominal hernia except by exclusion. There is usually a history of traumatism or peritonitis. The obstruction is generally in the small intestine, there is early fecal vomiting, no tenesmus, little or no meteorism and collapse occurs early. The extraabdominal hernias are readily diagnosed and when strangulated they give the ordinary symptoms described. *Volvulus* may sometimes be recognized as a painful mass in the left iliac fossa, with intense remissive pain and marked early tympanites. It is comparatively rare. *Kinks* usually produce chronic intestinal stasis but may sometimes completely close the lumen of the gut and produce the symptoms of occlusion. There will, however, be a history of chronic constipation with autointoxication lasting for many months. Any part of the intestine may be bound down and kinked, and a variety of symptoms referable to the intestinal tract are thus produced. *Intestinal paresis* is readily diagnosed by the associated paralysis. *Cicatricial stricture* gives a history of duodenal, syphilitic, typhoid, or tubercular ulcer. The diagnosis of intestinal occlusion is simple after feculent vomiting has set in. Before this, the pain and abdominal distention may be mistaken for appen-

Extensive protruding internal hemorrhoids. (From Gant's "Constipation.")

dicitis or peritonitis, but these diseases are accompanied by fever and chills. Hepatic colic with constipation may simulate occlusion. The remission of pain, the jaundice, light-colored stools, location of colic, and the history, will distinguish the two.

Treatment.—The treatment is purely surgical and should be undertaken as soon as the diagnosis of intestinal occlusion is established. Morphine and atropia may be given in the interim to relieve pain, and gastric irritation may be allayed by lavage, but nothing will relieve the obstruction except an operation. After collapse has occurred the case is hopeless.

HEMORRHOIDS

Hemorrhoidal Varix

Etiology.—Hemorrhoids occur frequently in persons past maturity owing to the greater tendency to venous stasis and the weakening of the venous walls, which permit their dilatation. They are mostly internal piles which protrude through the relaxed sphincter and in most cases are due to pressure upon the hemorrhoidal veins by feces that are retained in the rectum. The underlying cause is that of rectal constipation. Stricture, and tumors of the rectum or of adjacent organs, which press upon the hemorrhoidal veins, may also cause hemorrhoidal varix.

Symptoms.—*External piles* are seldom sufficiently distressing to require medical attention. They appear as tumors, ranging in size from a pea to a marble, situated outside of the anal sphincter. When inflamed or eroded by the friction of the feces, or by scratching where there is an accompanying pruritus, the surface is reddened or ulcerated, and painful. In rare instances there will be a hemorrhage; more often there is pruritus and eczema around the tumor, and the skin becomes strongly pigmented and infiltrated. *Internal piles* in the aged generally protrude from the anus after defecation and sometimes they remain permanently outside where the sphincter is lax. If returned within the rectum a slight strain will force them out again. They are readily recognized by their bluish appearance, doughy feel and motility under the mucous membrane which covers them. They are frequently inflamed,

excoriated or ulcerated from the irritation of the feces, and when in this condition they are painful and bleed readily. There is often an itching eczematous area around them, the skin becomes anesthetic, infiltrated and pigmented and may become ulcerated. If the sphincter ani has retained its tonicity an internal hemorrhoid which had been forced out may become strangulated, the enclosed blood stagnates and coagulates there and the tumor will be converted into a cystic mass, or may become gangrenous. Defecation may cause an inflammation about the base of the pile, and this periphlebitis, extending to the interior, produces a phlebitis. A proctitis, periproctitis, rectal abscess, anal fissures and fistulæ are occasional complications. The pain is usually not severe unless inflammation, erosion or ulceration occurs. When hemorrhage occurs it almost always accompanies defecation. In these cases there is generally a voluntary constipation, the patient fearing that defecation will produce pain or force out the pile. There may be tenesmus caused by irritation of the rectal wall, and there is sometimes a vesical irritation. In rare cases the pile is situated in the upper part of the rectum and is there more liable to become inflamed and to bleed. The cause or source of the hemorrhage may be a puzzle until an examination with the finger or proctoscope is made. It is hardly possible to mistake hemorrhoids for any other condition. Rectal and anal ulcers may bleed but do not present tumors. A carcinoma of the rectum is painful and rapid in its course; a polypoid bleeding growth does not have the color or consistency of the pile and breaks down upon slight friction.

Treatment.—Hemorrhoids in the aged are usually less distressing than in earlier life and may exist for years before a local pruritus, eczema or inflammation attracts the patient's attention to them. As soon as piles cause distress the annoying symptoms should be treated. For the pruritus we can follow the treatment suggested in the chapter on Senile pruritus and the eczema is to be treated as recommended in the chapter on this disease. Where there is an erosion or ulceration upon, or in the vicinity of a hemorrhoid, the surface should be covered with equal parts of subnitrate of bismuth and aristol over which a thick layer of unguentum petrolatum is to be applied in order to protect the lesion from the irritation produced by the fecal

discharges. If there is much pain in the pile a 2 per cent. cocaine ointment, using a lanoline base, should be used. This will generally give immediate relief, but if after two or three applications, the relief is not permanent the extract of belladonna should be substituted for the cocaine. Inflammation is best treated by applications of ice water (not ice), and, if there is hemorrhage, adrenalin in 1/10-per cent. solution will check it. Astringents will also check hemorrhage but they may produce a contraction of the anal sphincter and constrict the pile or, contracting the rectal wall, cause constipation. If operative procedure becomes necessary the choice of operation must be left to the surgeon.

BILIARY OBSTRUCTION

Etiology.—Partial biliary obstruction occurs generally from impaction of gall-stones or inspissated bile in some part of the duct. Other causes of biliary obstruction are impaction by concretions or parasites, inflammation of the duct or of the duodenum about the mouth of the common duct, contraction following angiocholitis, growths in the ducts, or pressure upon the duct by a growth in neighboring tissues; aneurysm, fecal impaction or bands of adhesions. It is often impossible to determine the cause of the obstruction, but other causes than impaction by gall-stones or inspissated bile are rare. An *angiocholitis* may occur as an extension of an inflammation of the duodenum, or may be due to irritation following the passage of a gall-stone. In most cases it is caused by the invasion of microorganisms, the colon bacilli, streptococci and staphylococci being generally found in the bile passages and in the bile of patients. In angiocholitis the mucous membrane is thickened and covered with thick tenaceous mucus, and if due to infection the secretion is mucopurulent. The swelling of the membrane usually produces a partial occlusion, but it may entirely obliterate the lumen of the duct.

There is no direct method of diagnosing angiocholitis nor can a presumptive diagnosis be made before jaundice appears. If symptoms of gastroenteritis precede the jaundice, there has probably been an extension of the inflammation into the duct. In this case there is a slowly increasing jaundice. Gradually

all the symptoms improve and the jaundice will dissappear at the same time. If there is fever, and later, a hepatitis with swelling and pain on pressure, the angiocholitis is due to infection. The symptoms are those of acute septic infection associated with jaundice and clay-colored stools.

Contraction of the duct may produce the symptoms of biliary obstruction. The diagnosis rests upon the history of a preceding angiocholitis, the former giving no pathognomonic symptoms.

Growths cannot be positively diagnosed unless they can be felt. Cancer gives the distinctive symptoms of such neoplasms, pain, and tenderness, the presence of a growth, rapid emaciation, progressive weakness, cachexia, and the involvement of neighboring tissues and lymph glands. *Aneurysm* of the aorta, hepatic or mesenteric arteries usually give distinctive symptoms. These conditions are, however, very rare in the aged and still rarer is occlusion produced by fibrous *bands of adhesion* resulting from peritonitis. They produce complete and rapid occlusion with profound systemic disturbance for the relief of which surgical interference is generally necessary. *Fecal impaction* is rarely massive enough to occlude the gall-ducts, and when it does occur it is usually relieved in a few days. Obstruction due to *gall-stones* or *concretions* is usually accompanied by the symptoms of gall-stones. These are paroxysmal pain and cramps with jaundice and intestinal disturbance due to deficiency of bile. In some cases it may be necessary to make the diagnosis by exclusion.

Symptoms.—The symptoms of biliary obstruction depend in part upon the location, and in part upon the degree of occlusion. The most prominent symptoms are jaundice and clay-colored stools, but if the cystic duct is occluded, both may be absent. The obstruction may be in the hepatic, cystic or common duct. If in the hepatic duct we must exclude gall-stones. If in the cystic duct the bile may flow from the liver through the hepatic and common ducts to the duodenum, thereby preventing retention of bile with the consequent jaundice and clay-colored stools. In such case the gall-bladder is unable to discharge its contents and these may cause inflammation or dilatation, or else calcareous or atrophic degeneration. Dilatation is rare in the aged as there is generally atrophy

of the mucous membrane and of the glands. If the glands are still active, the gall-bladder may become distended with mucus and bile, and where the abdominal walls are thin, the viscus can be felt as a pouch below and to the right of the sternum. Inflammation occurs occasionally, and almost always as a result of infection. A simple catarrhal cholecystitis may occur through extension of an angiocholitis, or from an irritation produced by the gall-stones contained in the gall-bladder, especially may this happen after rough manipulation of the distended organ. The only symptom pointing to catarrhal cholecystitis is pain and tenderness over the region of the gall-bladder. In infectious cholecystitis there are, in addition, the constitutional symptoms of septic infection, chills, fever, nausea, vomiting, distention of the abdomen, etc. In these cases surgical measures are required to determine the exact condition and to relieve the distended organ. In most cases of obstruction of the cystic duct, the gall-bladder undergoes calcareous and atrophic degeneration, the contained gall-stones becoming encapsulated. If the hepatic duct is obstructed, jaundice and light-colored stools appear and the liver becomes congested but the gall-bladder is not affected. If the obstruction is in the common duct, however, the gall-bladder becomes distended with bile that had been dammed back from the point of obstruction. Clay-colored or light-colored stools containing undigested fat indicate a diminution in the bile supply to the intestines. If this occurs without jaundice the fault lies in the liver, which is not elaborating sufficient bile, while a fairly dark stool with jaundice points to obstruction of some of the bile ducts in the liver. The stools in biliary obstruction are pasty and foul smelling. Sometimes there is constipation then again diarrhea as soon as intestinal decomposition causes irritation of the bowel.

The most important symptom of biliary obstruction is jaundice. The toxemic jaundice is readily differentiated by the presence of an acute infection, or of arsenic or phosphorus poisoning, in these cases the jaundice is not severe, the stools are bile-stained and the accompanying symptoms of obstructive jaundice, pruritus, sweating and bradycardia are not marked. Indeed, they may be absent.

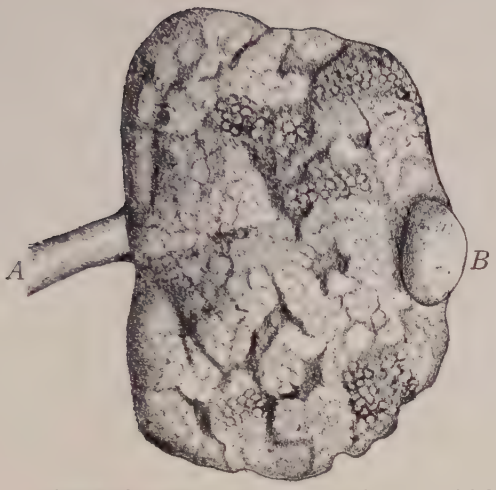
The jaundice due to obstruction of some of the bile ducts

in the liver may be mistaken for the jaundice of an infectious disease, as the stools are bile-stained and all the other symptoms are mild, but there are no symptoms of the infectious disease and bile pigment can be found in the urine. Jaundice may occur in chronic hepatitis, cirrhosis, cancer and other pathological states of the liver, but it is a late symptom of these diseases.

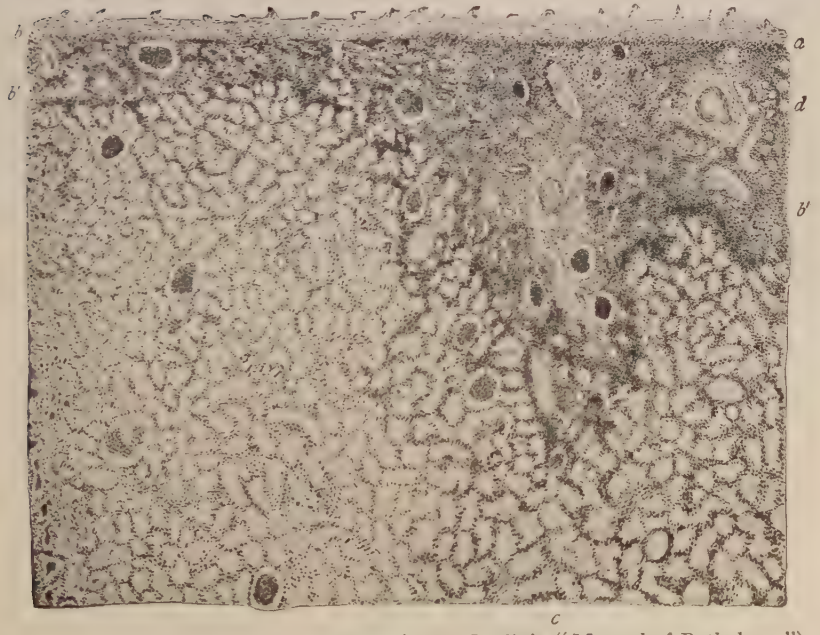
Treatment.—The treatment of biliary obstruction depends upon the cause. The treatment for cholelithiasis is given elsewhere. In catarrhal angiocholitis the alkaline mineral waters should be used, and sodium salicylate may be given in 5-grain doses combined with 1 dram of sodium phosphate every four hours. In all cases, whatever the cause may be, sodium choleate should be given in 3-grain doses after each meal. Potassium bicarbonate will increase the fluidity of the bile. Calomel in 1/10-grain doses every two hours will increase the activity of the liver, but in most cases the fault does not lie in this organ, therefore, hepatic stimulants are contraindicated. The attending symptoms, especially the often distressing pruritus, can be temporarily relieved by washing the body with a 1-per cent. solution of cocaine. In some cases a 2-per cent. solution of carbolic acid in oil will help, in others hot water or cold water will relieve the itching.

CHRONIC INTERSTITIAL NEPHRITIS

Chronic interstitial nephritis is the most frequent of the renal affections found in the aged. It is, however, not as frequent as the reports of pathologists would indicate, for pathologists still call the normal senile contracted kidney interstitial nephritis, and physicians still diagnose every persistent albuminuria as Bright's disease, especially if there are concomitant nervous symptoms. Walsh has pointed out that there is a physiological increase of connective-tissue growth between the apices of the pyramids going on from birth throughout life. This hyperplasia is most marked in old age when arteriosclerotic nutritional changes cause atrophy of other renal tissue. Faulty nomenclature is partly responsible for the confusion in diagnosis, since several distinct conditions are included under this term while the same condition has received several names. Chronic



Kidney Showing Advanced Chronic Interstitial Nephritis. (Natural size.) *A*. Ureter. *B*. Small cyst just under capsule. The irregularly lobulated, coarsely and finely granular surface is well shown. (From Coplin's "Manual of Pathology.")



Kidney, chronic interstitial nephritis. (From Coplin's "Manual of Pathology.")

interstitial nephritis, renal cirrhosis, sclerosis of the kidney, granular kidney, gouty kidney, contracted kidney, atrophic kidney, are all names used for this disease, while the same term is also applied to (1) a secondary condition following parenchymatous nephritis called also small white kidney, (2) to a primary pathological degenerative process, and (3) to the normal senile degeneration, generally due to renal arteriosclerosis. The last of these is the normal senile contracted kidney, the "rein sénile" of the French. The term chronic interstitial nephritis is here applied to signify a primary pathological degeneration, or perversion of the normal degeneration and not to the secondary involvement of interstitial tissue following parenchymatous nephritis.

Etiology.—The usual cause of chronic interstitial nephritis in the aged is excessive work imposed upon the physiological contracted kidney. The senile kidney cannot eliminate waste material as rapidly nor as actively as before and it is forced to increased activity whenever such material is produced in excess —as in excessive ingestion of food, especially of meat—or whenever waste is retained in excess, as in constipation, and when abnormal material must be eliminated such as lead, iodine, mercury, or the products of imperfect metabolism, etc. Any cause responsible for an increase in any of the normal constituents, or for the production of abnormal ingredients in the urine, is also the cause of excessive activity or irritation of the kidney and consequent degeneration. We, therefore, find it after prolonged physical or mental labors, indiscretions in food or drink, in gout, chronic rheumatism, diabetes and other conditions due to impaired or perverted metabolism. The toxins of infectious diseases cause a parenchymatous, rarely an interstitial degeneration although the latter may follow as a secondary affection through extension of the degeneration.

Pathology.—The kidney of interstitial nephritis resembles the normal contracted kidney in being small, rough, dense, dark red in color, granular and having a closely adherent capsule. There is an atrophy of the cortex, while the region of the pyramids exhibits a hyperplasia of connective tissue. The difference between the normal senile kidney and the kidney of interstitial nephritis is readily seen under the microscope. In the latter condition there are found hyaline and fatty degenera-

tion and cloudy swelling of the tufts, capillary vessels, and between the loops, the tubules are filled with casts and granular matter and some of the smaller vessels and glomeruli are destroyed. These degenerative changes are not found in the normal senile contracted kidney.

Symptoms.—It is impossible to distinguish between the early symptoms of chronic interstitial nephritis and the normal senile kidney. A more or less persistent trace of albumin is present in both and the early nervous and visceral symptoms may be due to the senile degeneration of the organs. The diagnosis must be made by carefully examining the urine. In nephritis the quantity is increased and the specific gravity is lower than normal. We must remember that the normal output in the aged is from 1000 to 1200 c.c. in the male and from 900 to 1000 c.c. in the female and what would be normal in maturity is a polyuria in old age. The specific gravity in interstitial nephritis is sometimes as low as 1.01 or even less. The finding of a single hyaline, fatty or granular cast determines the diagnosis and this will be confirmed by other symptoms. The patient must get up at night to empty the bladder. The aged usually get up once or twice a night for this purpose if they have a dilatation of the bladder but if one gets up several times at night it points to nephritis. Cardiac hypertrophy and high blood pressure are constant attendants. A persistent high blood pressure without arteriosclerosis is almost pathognomonic of this disease. Edema of the ankles may occur but this is rarely extensive until the heart is seriously involved. In many cases there are intermittent severe headaches, sometimes hemicrania, often insomnia and restlessness. Dyspnea and asthmatic attacks may occur. Later gastric symptoms, anorexia, indigestion and irregular bowel action are noticed. The skin becomes dry and there may be pruritus or eczema. Nervous symptoms appear later, such as tinnitus, disorders of sight, muscle twitching, cramps, etc. Diffuse retinitis and retinal hemorrhages, which occur frequently in younger individuals, are infrequent in the aged. The uremic convulsions which generally appear toward the termination of this disease in earlier life occur rarely in the old, the patient usually succumbing to an intercurrent disease, such as pneumonia, pulmonary edema or heart disease.

Diagnosis.—It is important to differentiate between the senile contracted kidney and the kidney of chronic interstitial nephritis. In the normal senile kidney the amount of urine is diminished, the specific gravity is but slightly if at all lowered, the urates are but slightly decreased and there are no casts. If there are cardiac, nervous or other symptoms, each symptom must be traced to its source and cause. If there is frequent urination at night we must look for a dilated bladder and enlarged prostate. High blood pressure may be due to arteriosclerosis. The headache, gastric disorders, and nervous symptoms must be considered one by one and their cause determined.

The gouty kidney, which gives symptoms of interstitial nephritis, can be diagnosed by other symptoms of gout. The secondary contracted kidney or small white kidney follows chronic parenchymatous nephritis which gives pronounced symptoms. It must be remembered that in all cases of nephritis the diagnosis depends primarily upon the urinary analysis, other symptoms being merely corroborative. If after repeated examination no casts appear at any time we can exclude nephritis. The primary interstitial nephritis has few casts, these are chiefly hyaline, and has but a scanty sediment. Other forms of nephritis have numerous casts and an abundant sediment. In both the primary and secondary interstitial nephritis the quantity of urine is increased but the specific gravity of the latter is but slightly reduced, while in the former it is very low. The presence of albumin does not necessarily imply nephritis, nor does its absence exclude this disease. It is always present in abundance in the parenchymatous form and it is present in small quantities in the secondary interstitial form. It may, however, be absent for some time in the primary form while a trace may persist in the normal senile kidney.

Treatment.—Degenerated tissue cannot be restored. The most we can hope to do is to avoid everything that causes or hastens the degeneration and to relieve symptoms or secondary conditions by drug medications. Where the causative condition is controllable we can sometimes expect an improvement in the condition of the kidneys, as may be seen in the gouty kidney under the treatment for gout. The general treatment of the senile kidney is hygienic and dietetic. The dietetic regulation is the most important and often the most

difficult factor in the treatment of senile cases. The patient should have a varied diet including all the food elements required for nutrition and in sufficient quantity to maintain normal weight. From this diet he must exclude as far as possible those substances that give the kidney excessive work, and those that would cause indigestion or constipation. The most important substances to be excluded or diminished in quantity are proteids, salt, alcohol and an excessive amount of fluid. Meat should be taken sparingly and omitted for several days at a time. The least harmful are the light meats, chicken, game and bacon. Broths are as injurious as meats. Vegetables except legumes, cereals and other farinaceous foods, fruits, fish and shell fish are admissible. Coffee and tea should be used sparingly but milk and buttermilk may be taken freely. The amount of salt should be diminished but a salt-free diet is inadvisable unless there is edema, an infrequent contingency in the aged. Alcohol should be forbidden unless the patient is accustomed to it, when the quantity should be gradually reduced. Alkaline mineral waters free from sodium chloride, preferably the natural lithia waters, are serviceable to prevent the formation of fibrinous plugs in the tubules. The hygienic regulations are a strict enforcement of the ordinary rules of health as applied to the invalid. Mental and physical fatigue should be avoided. Strong emotions and prolonged worry are detrimental, while mild pleasurable mental and physical stimuli are beneficial. Moderate exercise, stopping short of fatigue, should be taken, and warm baths are beneficial.

Drug medication is indicated as soon as distressing symptoms arise or whenever there is danger of grave complications, or for the treatment of the latter. For anorexia the simple bitters or orexin can be used. The bowels must be kept open and for this purpose aloin combined with the bile salts is indicated. The bile salts aid in preventing intestinal decomposition. Iron is frequently recommended in this disease, but in the aged where there is high arterial tension it is contraindicated. When the blood pressure is very high and there is danger of cerebral anemia or venous stasis the nitrites must be employed, using preferably, the 1-per cent. spirit of glonoin in 1-minim doses every three hours until the face becomes flushed and remains flushed for a few minutes. It should then be discontinued.

Diuretics are rarely indicated unless the degeneration involves the parenchyma and the amount of urine is markedly diminished. This, however, rarely happens. When diuretics become necessary, renal irritants, including the essential oils and oleoresins, should be avoided. In such case the sodium or potassium nitrate should be employed. In senile cases we must bear in mind not only the condition of the kidneys but the condition of the whole degenerate organism.

UROLITHIASIS

Renal and vesical calculi are frequent, the vesical calculus almost invariably originating as a renal concretion. The two forms of urolithiasis will be described separately.

Renal Calculus

Renal calculi occur frequently, sometimes without giving any symptoms of their presence. When minutely small they pass away with the urine unnoticed, forming an insoluble sediment; when larger they produce a local irritation in the ureters, bladder and urethra and pass away as gravel, or coarser sediment. Still larger concretions pass through the ureters with difficulty and produce the painful symptom-complex of renal colic. If too large to pass through the ureter the renal calculus becomes impacted or imbedded in the pelvis of the kidney. The concretion generally consists of uric acid, sometimes of urates or phosphates, occasionally of calcium or carbonate oxalate, cystin, xanthin, fibrin, etc. The experiments of Ebstein and Nicolaier have shown that the structure of the renal calculus is an albuminous framework filled with calcareous material, deposited either in concentric layers, scales or threads, or else as irregular crystals. The growth of the calculus keeps pace with the growth of the framework. The nucleus of the stone may be a microscopic crystal, pus, blood, pigment, fat, fibrin, cystin, tube cast or other urinary constituent, microorganisms or parasitic ovum. The framework is derived from an inflammatory process in the kidney, the calcareous material from the urine.

Etiology.—Two etiological factors are necessary for the production of renal calculi; one which will cause the production of

the framework, the other which will cause a change in the character of the urine. Anything which will produce an irritation of the kidney will cause a mild catarrhal inflammation with secretion of mucus from which the framework material is obtained. A highly acid urine or any other renal irritant may do this. The most frequent change in the character of the urine is an excess of urea or uric acid, and we find, consequently, that uric acid calculi occur in the aged more frequently in connection with gout. Drinking excessive quantities of earthy mineral waters predisposes to phosphatic calculi. The derivation of the ammonium-magnesium phosphate in phosphatic renal calculi is not clear, as the ammonia element is produced in the decomposition of urine and such decomposition in the kidneys is generally due to bacterial infection. When this occurs pyelitis usually results, yet calculi of triple phosphates have been found in the kidney without any other kidney involvement.

Symptoms.—In some cases there are no symptoms to indicate the presence of a renal calculus. In most cases, if the calculus remains in the kidney, there is a dull ache in the lumbar region, with occasional pains shooting downward and forward toward the bladder, or down to the thighs. The pain is aggravated by anything that would disturb the position of the stone, as jolting, horseback riding, jumping, etc. This is occasionally followed by hematuria and pyuria. Septic symptoms may arise. Small calculi generally pass from the kidney to the bladder, producing during their passage through the ureter the symptoms of renal colic. There is a sudden intense pain extending from the kidney to the testicles or labia, especially severe at the point where the stone is momentarily lodged in the ureter. There is at the same time a sharp pain at the end of the penis, and the testicle on the affected side is retracted. There is also a constant desire to urinate, but only a few drops are passed at a time, and the urine is then generally blood-stained. The usual concomitants of shock are present, namely, intense pain, an anxious, pale, pinched countenance, covered with cold perspiration, nausea and vomiting, small pulse, slight elevation of temperature and collapse. The symptoms abate as soon as the calculus has entered the bladder, the time of passage varying, generally from one hour to a day. The aching pain across

Heberden's nodes. (Courtesy of S. Epstein, M. D., New York.)

the back may continue for two or three days, but is gradually diminishing in severity. Immediately after the passage of the stone into the bladder there is a copious flow of urine which may contain albumin, casts and blood. Occasionally a calculus becomes impacted in some part of the ureter. In such case the colicky pains will persist for days with a gradually diminishing intensity. A hydronephrosis follows, but if the other kidney is healthy this will give no symptoms until the excessive work imposed upon the healthy kidney causes its degeneration. Pyonephrosis and pyelitis may follow septic infection.

The urine is acid if there is a uric acid calculus and alkaline if there is a phosphatic or oxalic concretion.

Treatment.—A stone impacted in a ureter or so situated in the kidney as to interfere with the discharge of the urine, thereby producing much distress, or causing inflammation, must be removed by surgical means. If there is but distress without inflammation and no interference with the excretion of urine, medicinal measures may first be tried. The best drug for dissolving uric acid calculi is piperazine. This should be given with alkaline waters, acetate or citrate of potash, citrate of lithia, benzoate of soda or any other alkaline salt which will render the urine alkaline. If the urine is alkaline, pointing to phosphatic stone, the first indication is to render it acid by benzoic, boracic, or the mineral acids. Theoretically this should dissolve the stones. Prolonged acid medication has been followed by renal colic. It would appear that the acid diminishes the size of the calculus and enables it to pass through the ureter.

In the vast majority of cases medical aid is first sought when renal colic appears. The only medical aid possible is to relieve the pain by hypodermics of morphine or inhalations of chloroform. Hot applications to the abdomen and warm baths may give momentary relief, but unless the calculus is small and passes readily and rapidly through the ureters, the narcotics are indispensable. Morphine can be given in 1/4-grain doses combined with 1/100 grain of atropine. Hematuria is rarely severe enough to require treatment. If pyuria is present it must be treated as due to pyelitis. When surgical intervention is necessary the character of the operation must be left to the surgeon.

Vesical Calculus

Vesical calculus occurs more frequently in old age than in earlier life and mostly in men who have a hypertrophied prostate. Many cases originate as a renal calculus which has passed into the bladder, either as gravel without colic, or as a larger concretion. The bladder being generally dilated in the aged, the base forms a pouch behind the enlarged prostate and the gravel, or stone, drops into this pouch and forms the nucleus for the vesical stone. The structure is the same as in renal calculus, mucus supplying the albuminous material for the framework and the decomposing urine furnishing the ammonia which combines with the earthy phosphates, the latter being precipitated in alkaline urine. The nucleus, if coming from the kidney, is usually a small uric acid calculus; when originating in the bladder, it may be any constituent of the urine which is liable to be precipitated in an insoluble form, or else it is epithelial débris, fibrin, cystin, etc. It has been suggested that microorganisms are responsible for vesical and renal calculi but these appear to cause only urinary decomposition with production of ammonia. The production of calculi is simply the result of chemical and mechanical processes and of a pathological separation of albuminous matter from the mucus which forms the framework of the stone.

Symptoms.—The passage of a renal calculus with the attending renal colic and the certainty that the stone had not passed through the urethra is conclusive evidence of the presence of a calculus in the bladder. A calculus which produces colic when passing through the ureter will also cause intense pain when passing through the urethra. The symptoms of vesical calculus in the aged when the stone is lodged at the base of the bladder behind a hypertrophied prostate, are often so vague that a positive diagnosis cannot be made without instrumental examination or radiography. The pathognomonic symptom of vesical calculus in earlier life, a sudden blocking of the urethra while urinating, does not appear when the stone lies behind the prostate. In most cases there is a dull ache in the perineum, aggravated by jolting, long marches or any motion which would disturb the stone. Prolonged sitting which aggravates the ache of an enlarged prostate has no such effect

upon a vesical calculus. Hematuria may be present and this symptom will simulate acute cystitis. In the latter disease there are vesical and rectal tenesmus and frequent urination, the urine is mixed with mucus, pus, and epithelium, all of which are mild or absent in vesical calculus. The stone can sometimes be felt by the finger through the rectum, its hard consistency distinguishing it from growths and a hypertrophied prostate. Vesical exploration by means of a metallic sound and of a cystoscope gives the most certain information but in hypertrophied prostate it is sometimes difficult to so manipulate the instruments as to bring the portion of the bladder situated behind the prostate within view or touch. In rare cases the stone may be imbedded in one of the vesical pouches and the pain will then be located over the location of the stone. In such case the stone will produce a chronic cystitis but unless it is dislodged by a jolt a positive diagnosis can be made only by instrumental exploration or X-ray. When the sound and cystoscope fail to reveal the presence of a stone and there is any difficulty in reaching all parts of the viscus, a radiograph is necessary to clear up the diagnosis. This course is better than to place the patient under an anesthetic and subject him to rough exploratory instrumental manipulation.

Treatment.—Internal medication, except for the relief of symptoms and the prevention of urinary decomposition, is useless. Injection into the bladder of a very weak solution of dilute hydrochloric or nitric acid has been advocated, but a weak solution has no effect upon phosphatic stone, while a stronger solution will produce an acute cystitis. We must remember that, while the nucleus may be a uric acid concretion from the kidney, the vesical deposit almost always consists of phosphates and the uric acid solvents are ineffectual. The only radical treatment is surgical, the preferable operation being lithotrity. In many cases where an operation for removal of stone is necessary, a hypertrophied prostate can be removed at the same time. When a condition exists making it advisable to perform a prostatectomy at the same time, the preferable operation is the suprapubic cystotomy advocated by Lilienthal. This should be performed under local anesthesia.

In the ordinary **chronic cystitis** the primary cause, if persistent, must be removed. If this cannot be done, measures

to correct decomposition of the urine and irritability of the bladder must be employed and continued. After the turbidity has cleared up salol should be used in small doses, 1 grain two or three times a day, to prevent its return. Irrigation is useful where there is pus or persistent mucus in the urine. If pus is present the silver salts are preferable but if there is only mucus, sodium borate and sodium sulphite should be used in the proportion of 1 dram to 4 ounces of warm water once daily. Hot applications over the bladder and hot enemata will usually relieve the pain which is rarely severe unless there is ulceration. Hyoscyamus in 5-minim doses of the fluid extract is probably the most effective drug to relieve pain and irritability in these cases.

Acute cystitis is infrequent in the aged, except as a mild infection occasioned by the introduction of a dirty catheter or a non-sterile irrigating liquid, while a mild irritation may be produced by some irritating abnormal ingredient of the urine. In either case it soon becomes chronic. An active acute inflammation is extremely rare and does not differ from the same disease in maturity.

SENILE METRORRHAGIA

Metrorrhagia is a symptom of various uterine disorders. Occurring during the menopause it is usually the menstrual flow coming on at irregular intervals. It may, however, be due to an endometritis, prolapsed uterus or to a growth. Endometritis and prolapse give clearly defined symptoms and the metrorrhagia accompanying these conditions is readily controlled by styptics. Metrorrhagia due to fibroids, polyps or cancer is more persistent and continues after the completion of the menopause. In the case of fibroids the flow usually diminishes as the senile involution of the organ proceeds and it may cease completely. Polyps usually have a copious flow coming on in spurts or there may be a continous dribble. It generally diminishes during the menopause and may cease altogether. The flow that accompanies malignant disease begins as an insignificant watery, scanty discharge having a pinkish tinge and slight or no odor. This discharge may exist for months before any attention is paid to it. It gradually

Spondylitis Deformans. Regular Contour. *X*-ray shows ossification of inter vertebral articulations. (Courtesy of S. Epstein, M. D., New York.)

becomes darker and more copious and begins to have a fetid odor, which increases in intensity until it becomes intolerable. When this condition is reached the only question of diagnosis is between cancer and senile metritis (see Senile Metritis). In the early stage the effect of local treatment will usually suffice to distinguish the metrorrhagia of malignant disease from other forms of metrorrhagia. The discharge due to malignant disease will persist in spite of the use of styptics and astringents, their action lasting but a few minutes or hours. In other forms of metrorrhagia the flow is controlled temporarily and often permanently under local treatment.

A metrorrhagia beginning after the completion of the menopause is almost invariably due to malignant disease. Other causes are the hemorrhagic form of senile metritis, cardiac disease and traumatism, but these are extremely rare. The metrorrhagia in malignant disease sometimes begins as a scanty, thin, yellowish discharge, slowly becoming pinkish then darker until it is dark red or, if mixed with pus, a dirty red. At the same time it becomes thicker, more copious and continuous and assumes a fetid odor. In some of these cases the discharge is yellowish or grayish and contains drops or streaks of blood. In other cases the discharge is slight for a time, suddenly becoming copious or appearing abundantly for a few hours then diminishing again, these gushes coming on at irregular intervals. It is hardly necessary here to give the other symptoms and signs of uterine cancer. The pain, sensitiveness on pressure, enlargement of the organ and general cachexia, all point to malignant disease, but the diagnosis must be confirmed by an examination of a curette scraping.

The treatment of senile metrorrhagia depends upon its cause. If it is simply an irregular menstrual flow nothing need be done, but absolute rest may be necessary. Astringent solutions will generally avail in the case of endometritis and prolapse. These, and ergot internally in 1/2-dram doses, will generally temporarily control the loss of blood from fibroids and polyps; surgical intervention, however, may be necessary to remove the cause. In all cases where there is an exhausting loss, hot douches should be given and if these do not suffice to control the flow astringents like tannic acid, perchloride of iron or zinc sulphate should be added. As a last resort packing of the uterus may

be tried but occasions for this are extremely rare. The most effective means to destroy the fetor of cancerous discharges is a douche containing a tablespoonful of a 3-per cent. solution of permanganate of potash to a pint of water. Hot douches have no effect in controlling hemorrhage in uterine cancer. If hemorrhage occurs, powerful astringents, such as Monsell's solution diluted 1 to 8, or the sulphate of iron and ammonium in 10-per cent. solution, will be required. The effect is, however, only temporary and the cure will depend upon the cure of the causative condition.

CHRONIC RHEUMATISM

This is a primary disease of middle and advanced age which, in its pathology, resembles the changes of senile arthrosclerosis.

Etiology.—In some cases there is a history of earlier attacks of acute articular or subacute rheumatism, but only in rare instances has either of these diseases immediately preceded chronic rheumatism. The basic etiological factor is unknown, but it occurs most frequently in those who are much exposed to cold and dampness and who are generally weakened by improper living and hard work. The disease is probably but a perversion of the ordinary senile processes in the joints brought on earlier than usual through some local causes.

Pathology.—The most marked anatomical changes are found in the articular cartilages which become roughened; and in the ligaments and tendons, which become thickened and hardened. The synovial membrane also thickens and the synovial fluid is usually diminished in quantity. The muscles atrophy from disuse and there are often evidences of senile changes in other tissues.

Symptoms.—The disease is slowly progressive with occasional acute exacerbations and often with long periods of remission. It begins as a dull ache in the affected joints, generally in the evening after the joints have been actively used during the day. They feel stiff and sore and may be swollen. The stiffness persists throughout the night and is relieved after slight active motion in the morning. Slowly and gradually the stiffness increases until finally the joint is completely and permanently anchylosed. It may take many years after the initial

symptoms appear before the final result is reached. Exacerbations with increased stiffness, pain and swelling, which lasts for several days, will occasionally occur. The joints most frequently affected are those most frequently used or exposed to deleterious influences, *i.e.*, the hands of manual laborers and the feet of those who walk much. In many cases one hand or one foot alone or a hand and a foot on the same side are affected. The large joints are seldom involved. The disease is frequently associated with senile changes in other tissues and these contribute their symptoms to the symptoms of the disease.

Diagnosis.—In the early stage of the disease it must be differentiated from the early stage of arthritis deformans and from senile arthrosclerosis. In multiple arthritis deformans a number of joints are affected and the disease is bilateral, osteophytes appear and the flexors are contracted. It is impossible to distinguish between chronic rheumatism and arthritis deformans in an early stage when only a single large joint is involved. Later, the presence or absence of flexion and deformity will determine the diagnosis. It is also important to differentiate between chronic rheumatism and arthrosclerosis and this can be determined by a single symptom. The pain and stiffness of the joint in chronic rheumatism are relieved after limbering up in the morning, while in arthrosclerosis there is no pain during rest and motion produces more and more pain. In gout a single joint is affected and there are paroxysmal attacks coming on at night. In gonorrheal rheumatism there is the history, the symptoms are more active and the disease is rare in the aged. Progressive muscular atrophy has been mistaken for chronic rheumatism, but in that disease there is little or no pain. The difficulty of motion in these cases is due to waste of muscle and not to joint stiffness, the apparent enlargement of the joints being due to the retraction of the wasted muscles.

Treatment.—Permanent arrest of the disease has followed a complete change in the mode of life of the patient with avoidance of exposure to cold and wet and residence in a dry warm climate. The iodides are sometimes beneficial, but more lasting results have followed hydrotherapy, electrotherapy and thermotherapy. In some cases hot applications, in others cold

applications seem to do better. The "baking" process has been followed by permanent relief and cures have been reported from the use of the high-frequency current. Massage and vibration frequently relieve the stiffness and have been found of temporary benefit even after complete anchylosis. General tonics must be employed and for this purpose nothing equals phosphorus and arsenic.

ARTHRITIS DEFORMANS

This disease, often erroneously called chronic rheumatism, is a primary progressive disease of the joints occurring during or after middle life.

Etiology.—The basic cause is uncertain. Two general theories are held, (1) that it is of nervous origin and (2) that it is a bacterial disease. Poncet claims it to be a tubercular affection of the joints, and Valentine found that 40 per cent. of cases of arthritis deformans had tuberculosis. The bacterial theory is based upon the fact that microorganisms have been found in the joints of cases that began with acute symptoms. As the disease is usually insidious in its advent, it is probable that, where cases begin with acute symptoms, those symptoms belong to an acute infectious disease, perhaps to acute articular rheumatism, with which the arthritis deformans has nothing in common except the one single symptom of pain in the joints. The neurotic theory is based upon (1) the similarity of the lesions to some spinal-cord lesions, (2) the frequent occurrence of dystrophies, (3) the influence of mental disturbances and emotions in its causation. Each of these can be controverted by the simple fact that they do not apply to the majority of cases. Damsch offers a toxin theory. Ord advances a theory that the disease is due to a lesion in the trophic centers of the cord. A further study of this disease, however, shows that the anatomical changes are identical with the normal senile joint changes, but they proceed faster and are carried further than in senile arthrosclerosis. The disease begins in the joints that have been most actively employed, generally the hands, followed by the ankles and feet, then the knees, wrists, elbows, shoulders, cervical spine, hips and lastly the dorsal spine. The flexor muscles, which are the ones that are the most

Spondylitis Deformans. Irregular Contour. Sideview. (Courtesy of S. Epstein, M. D., New York.)

actively employed, become permanently contracted, thereby producing the deformities which are pathognomonic of this disease. From these facts it would seem that the disease is but an early and exaggerated senile process. The pains are due to degeneration of the nerve terminals in the affected tissues.

The exciting causes are unknown. Every conceivable departure from a natural mode of life—the excessive use of amylaceous and saccharine food, exposure, exhaustion, sexual excesses, unhygienic surroundings, rapid temperature changes, etc., have been cited as possible exciting causes.

Pathology.—The articular cartilages become dry, fibrillated and wear away through attrition, leaving the bone exposed. The spongy portion of the bone wastes, the articular surfaces roughen and eburnation ensues as the result of friction. Osseous nodules, or a complete osseous ring may form about the articulating surface. The synovial membrane thickens and the sac becomes dry. Thickening and hardening of the ligaments and tendons and waste of the muscles contribute to the anatomical changes which cause the characteristic deformities marking the disease.

Symptoms.—The disease appears in three forms, complete or multiple, partial, and abortive.

The *multiple form* is the most frequent, and usually begins as a dull ache in a single joint of a finger or toe which later becomes swollen and painful to the touch or upon motion, while the joint becomes flexed. In the meantime the corresponding joint on the other side becomes affected. The affection spreads to other joints of the hand or foot and to the corresponding joints on the opposite side. The effusion is slight, never as extensive as in acute articular rheumatism, nor is the pain in the early stage of the disease severe. There may, however, be neuralgic pains, or the more persistent pains of neuritis, due to irritation or degeneration of the nerve terminals in the affected tissues. The disease is progressive, with frequent remissions, the relapses being generally more severe than the previous attacks and increasing the deformities. In an advanced stage of the disease the fingers turn toward the ulnar side, are flexed and may overlap. The wrists turn outward, the elbows are bent, the shoulders are fixed, with the

arms hanging down and the hips and knees are flexed. The amount of rigidity in different joints may vary, but the corresponding joints of the two sides are generally affected to the same extent.

Complete anchylosis is rare, true bony anchylosis occurring only in the spinal column. A famous example of complete rigidity of the joints was "the ossified man," who was on public exhibition for many years. Various skin disorders occasionally appear in connection with the disease. There may be pigmentation, bromidrosis, or local sweating, paresthesias, etc.

The acute form of rheumatic arthritis, which begins with symptoms resembling an acute articular rheumatism, does not occur in old age.

The *partial form*, also called the *monarticular type* is confined principally to one, or a few of the larger joints, while the smaller joints either escape entirely, or are but slightly affected. There is the same train of symptoms, beginning with tenderness, then effusion and pain with remissions and exacerbations, gradual stiffening of the joint, and deformity.

Morbus coxæ senilis is a form of partial arthritis deformans in which one hip, or, rarely, both hips are affected. The capsular ligament and ligamentum teres contract and other joint changes take place. The leg is apparently shortened and gradually becomes fixed in a bent position.

Spondylitis deformans is a rheumatic arthritis confined to the spinal column. It occasionally terminates in complete bony anchylosis. The *abortive form* of arthritis deformans usually begins in the distal joints of the fingers but rarely passes beyond them, though it may affect the toes. It is marked by the production of exostoses, called "Heberden's Nodes," ranging in size from a pin's head to a pea, which form on the sides and ends of the distal joints of the fingers. The changes in the joints are the same as in other types, but there is rarely a contraction of the flexor tendons or waste of muscle. In some cases the presence of the nodes is the only symptom.

Diagnosis.—In the early stage of arthritis deformans it is often difficult to distinguish it from other arthritic diseases. The acute form of the multiple type resembles, in its onset, acute or subacute articular rheumatism, but this form does not occur in the aged. The slow insidious advent, the absence of fever,

the involvement of the small joints, and the stationary character of the anatomical changes in the beginning of the disease will distinguish it from subacute rheumatism. Crepitation upon motion which is appreciable to a delicate touch, is often an early symptom of rheumatic arthritis. This is absent in subacute and chronic rheumatism. Chronic rheumatism is generally unilateral, and the affected joints are usually stiff and painful after prolonged rest. The stiffness and pain are diminished after motion, whereas motion increases the pain of rheumatic arthritis, resembling, in this respect, senile arthrosclerosis.

Gout attacks a single joint, generally of the big toe, the attack is paroxysmal, comes on at night and is much more severe than the attack of rheumatic arthritis.

Other arthritic diseases can be eliminated by the age, history, or by pathognomonic symptoms.

Treatment.—The disease is progressive, but the attacks of swelling and pain become gradually less frequent and less painful, while the rigidity proceeds until the patient is bedridden. The disease is incurable and, while temporary relief can be afforded during the acute exacerbations, no method of treatment has given permanent results. Drug treatment is useless except to relieve pain, when the salicylates or opiates may be given. Of the non-medicinal measures, hydrotherapy, electrotherapy, mechanotherapy and thermotherapy have been employed in various forms, some cases being temporarily relieved by one form of treatment, while in other cases the progress was apparently hastened. The most that can be expected from them is a prolonged remission with temporary lessened rigidity of the joints. In one case treatment at the hot sulphur springs at Aix la Bains was followed by a remission lasting two years. Hot baths and fomentations give temporary relief. The high-frequency current has been found beneficial in some cases and reports of apparent cure have followed the use of the X-ray with massage. Favorable reports have come from the hot-air treatment and Bier's hyperemia treatment.

Temporary relief from the deformity of the hands was obtained in one instance by immersing them in hot water for half an hour, then forcibly extending the fingers. The fingers remained extended for several days and motion was possible, but they soon began to resume their flexed position.

Of hygienic measures, mild exercise and the prevention of mental depression caused by the ill success of treatment are the most important. Gentle exercise is necessary, but fatigue should be avoided. Active exercise of the affected joint hastens the pathological changes, while no exercise will cause waste of muscle from non-use. Of psychic measures, change of surroundings and scene is the most important. The benefits derived from a trip to a watering place, or to medicinal springs are attributable, in great measure, to the change of surroundings, for the same mineral waters taken at home do not produce the same results. Other hygienic measures, such as a dry equable climate, the avoidance of surface chilling, nutritious dieting, etc., are self understood.

PAGET'S DISEASE

Etiology.—This rare disease of the bones occurs almost exclusively in advanced age. It consists of an increase in the volume of bone as seen in acromegaly and a softening of bone as in osteomalacia and it is supposed that the same causes producing these conditions, namely, disease of the hypophysis and of the thyroid, are responsible for the trophic changes in Paget's disease also. Numerous other causes, such as fatigue, exposure to cold or wet, traumatism, syphilis, cancer, etc., have been suggested. Some observers found spinal lesions in their cases, others failed to find any, but arteriosclerosis of the vessels supplying the affected bones is found in almost every case.

Pathology.—The anatomical changes in Paget's disease consist of waste of bone tissue in some places and hyperplasia in others. The Haversian canals in some localities are enlarged, in others, obliterated. There is no uniformity in location, degree or extent of these changes and all the bones may be affected, but the most pronounced changes are found in the tibiæ and femurs. Owing to the softening of these bones and to the downward pressure of the body upon them, they become curved and the neck and shaft of the femur form a right angle. The marrow is very vascular and the periosteum is thickened.

Symptoms.—The onset of the disease is insidious, often unnoticed until a change in the shape of the bone is observed.

Spondylitis Deformans. Irregular Contour. Backview. (Courtesy of S. Epstein, M. D., New York.)

In some cases there are vague pains and aches as in chronic rheumatism, occasionally there are paroxysmal sharp pains in the affected bones. Pain is sometimes present throughout the disease, in some cases aggravated upon walking, in some cases coming on in paroxysms, in other cases absent. When the spinal column is pressed upon, as may occur in the deformity that accompanies spinal osteitis deformans, or when a nerve is compressed the pain becomes increasingly severe. The most important symptoms are thickening and malformation of the bones, the character and extent of deformity depending upon the location of the affected bones and the amount of pressure to which they are subjected. For this reason the spine and lower extremities are most deformed, the increased curvature of the spine producing a change in the shape of the thorax.

The only disease which bears any marked resemblance to Paget's disease is osteomalacia, which is very rare in the aged. In osteomalacia all the bones of the body are affected, the curvature of the spine is very marked and the pelvis is deformed, while in Paget's disease the deformity is usually confined to the lower extremities. Pain is usually pronounced in osteomalacia and generally absent in Paget's disease. The bones are not increased in size in osteomalacia. Acromegaly is extremely rare after the sixtieth year and it does not affect the extremities.

The disease is incurable but it may last for ten or fifteen years before an intercurrent disease, generally bronchopneumonia, causes death.

There is no known method of treatment and the only thing that can be done is to treat the distressing symptoms. Phosphorus has been found of benefit in osteomalacia and it may relieve symptoms of Paget's disease, but no cure has yet been effected.

(Pseudo-Paget's disease is included under the first group.)

GOUT

"Gout is a clinical syndrome arising from defective assimilation of nitrogenized substances."[1]

Numerous theories have been advanced to explain the pathogenesis and nature of gout but no one is free from unanswerable

[1] Rathery, "Manuel des Maladies de la Nutrition."

criticism. Biurate of soda is deposited in the joints, but the origin of the uric acid is uncertain, some investigators believing it to be a product of incomplete metabolism, some think it a product of perverted metabolism, while others say it is the product of complete metabolism of purin-forming substances. Garrod's theory that uric acid is retained in the blood instead of being eliminated by the kidneys has now few supporters. He placed the primary fault into the kidneys. Another theory explains gout as due to a hyperproduction of uric acid, a minute quantity being normal to the individual. Ord ascribes the source of this hyperproduction to the products of degeneration of certain fibrous tissue; Murchison claims it to be a functional perversion of the liver whereby albuminoid material is converted into uric acid instead of into urea; Ebstein believes that the sources of normal production of uric acid are multiplied in gout. Another theory is that uric acid and the purins are not completely converted or destroyed owing to deficiency of deoxidizing ferments (oxydases) in the blood. Other theories ascribe the presence of an excessive amount of uric acid in the circulation to the changed condition of the blood, a uric-acid dyscrasia, the diminished alkalinity causing incomplete oxidation; others claim that increased alkalinity makes the blood a poorer solvent, therefore, a larger quantity of uric acid is thrown from its solution and deposited in the tissues, again that the tissues in which the biurate of soda is deposited are less alkaline than the blood, some think that where the circulation is slowest the salt is deposited, and finally, that certain tissues have an affinity for this salt. Other theories are based upon the chemical changes by which the biurate of soda is produced; that uric acid and thyminic acid are formed together from the nucleins and are in combination and that in gout more uric acid is formed; or that uric acid is derived from substances that do not form the thyminic acid, or that uric acid is precipitated as an insoluble biurate in the presence of glycocoll, or that the urates exist in two forms, a stable and but slightly soluble one and an instable and readily soluble one, that the latter is converted into the former, etc. The latest theory is that uric acid is produced in excess from certain proteids containing purin-forming bases and that it is the end result of the metabolism of such purins. Another theory which has many supporters ascribes gout to a faulty metabolism

of proteids through failure of the nervous system to properly regulate the process of metabolism.

These are but few of the many theories that have been advanced to explain the pathogenesis and nature of gout. There are arguments which cannot be controverted in some cases, while in other cases they are completely refuted.

Etiology.—In most cases there is an inherited gouty diathesis. In some there is another disorder of metabolism such as obesity, diabetes, etc., giving symptoms of gout in addition to its own symptoms and the disappearance of the other disorder relieves the symptoms of gout.

It is most frequently found in those using fermented liquors and in countries where wines, heavy beers and ales are consumed in large quantities. Its frequency among those who drink such liquors, and its rarity among drinkers of distilled liquors would seem to point to a fermentation product and not to the alcohol as the etiological factor in these cases. This would also explain the increase of the disease in America, keeping pace with the increase in the consumption of beer. It is probable that its frequency among lead workers, type founders, painters, etc., is due to the large quantities of beer and ale consumed by them to quench the intolerable thirst of chronic plumbism. In many cases of chronic lead, zinc or mercury poisoning or where opium, belladonna, iodides or nitrites have been used, there is more or less suppression of the secretions, with consequent excessive thirst, which is often quenched by alcoholic, or, more especially, fermented liquors. This will account for the frequent attacks of gout in such conditions. It may be, too, that these toxins interfere with metabolism and if combined with a gouty diathesis an attack will be produced.

Rich, highly seasoned food in an excessive amount is an etiological factor, such food being also rich in purin-forming material. Gout, however, often attacks those who are insufficiently nourished and the so-called poor man's gout does not differ from the gout of those who live idle lives, eat rich food and drink heavy wines. While excesses in food and drink, especially of nitrogenized foods and fermented liquors, are the principal etiological factors, yet any mental strain, sudden emotion, infectious disease, traumatism, excessive venery, or fault in the mode of life may bring on an attack in a person having the gouty diathesis.

Pathology.—The pathognomonic lesion is a deposit of biurate of soda in the affected joints. This begins just below the free surface of the articular cartilage and the deposit gradually increases, invading the joint structure and incrusting the cartilage with a layer of sodium biurate. Later the tendons become involved and a salt deposit is found upon them and sometimes upon the synovial membrane. The synovial fluid becomes thickened and may contain crystals of the salt. The salt frequently collects in small masses, called tophi, which surround the joint and may appear on the tendons a short distance from the joints. Tophi are also frequently found in the cartilage of the ear, occasionally in the cartilages of the nose and other cartilaginous structures, but rarely in muscle.

During an acute attack, the affected joint is inflamed. In most cases the disease begins in the first joint of the big toe, later involving the ankles, knees and lastly the joints of the fingers. Occasionally the fingers are first affected. The kidneys are sometimes involved, showing either the changes of nephritis or biurate of soda deposits.

Varieties.—Many forms and varieties of gout have been described, all can be placed under two heads, however, *i.e.*, regular and irregular gout, the latter usually called goutiness. Gout is a chronic condition, every case being chronic from its inception, and the term chronic gout in contradistinction to other forms of gout is a misnomer. The paroxysmal attacks called acute gout are incidents occurring in the course of the chronic disease, and cannot be considered an entity apart from the chronic condition any more than could the symptom-complex known as cardiac asthma, occurring after exertion in cardiac dilatation, be called a separate disease. Gout rarely begins with an acute attack. Premonitory symptoms showing the existence of a gouty condition usually appear days, weeks or months before the acute attack and there is almost always some discoverable cause for it. What is usually described as acute gout will be treated here as an acute attack of regular gout. Irregular or extra-articular gout, or goutiness, is applied to a number of ill-defined pathological lesions or functional perversions found in persons having a gouty diathesis. With the increase of goutiness there is an increasing tendency to ascribe to it any pathological condition for which no other etiological factor can be

Paget's Disease. Nouvelle Iconographie de la Salpetriere, May–June, 1905.

discovered. This variety of gout presents an acute phase, the retrocedent gout which immediately follows the acute attack of regular gout.

Symptoms.—In some cases of *regular gout* there are no marked symptoms until the onset of the acute attack, in other cases there are prodromal symptoms appearing a few days before the attack, while in some there are various functional impairments with occasional twinges in the small joints for weeks or months before an attack occurs. Between the attacks, the patient may feel in perfect health, or there may be functional impairments (which will be described under Irregular Gout) or twinges in the affected joints. Usually the patient does not notice the prodromal symptoms of the first attack, headache, loss of appetite, malaise and little aches and twinges in the small joints of the toes, and occasionally of the hands. Having once experienced an attack, however, he will quickly notice these premonitory signs and there will be restlessness and depression brought on by the anticipation and fear of the attack. The onset is ushered in with an intense pain coming on suddenly or rapidly and generally at night, and in most cases involving the big toe. The joint becomes red, swollen, intensely painful, and tender to the touch, and there are the usual concomitants of fever, rapid pulse, dry skin, thirst, headache, mental excitement and general malaise. The symptoms abate somewhat during the day, becoming worse at night. After the third or fourth day the symptoms become gradually milder and the arthritic inflammation disappears in about ten days.

The urine during an attack is scanty and strongly acid, depositing urates upon standing. The amount of uric acid eliminated during the attack varies; occasionally none is found, at other times it may be present in excess. After the attack, the quantity of urine is increased and its specific gravity is diminished but it remains hyperacid and there is a large excess of uric acid for several days.

The acute attack of regular gout is often followed by an acute attack of irregular or retrocedent gout.

After several attacks, tophi form in the joints and in other places, the attacks becoming less frequent and in the aged less severe.

The protean character of *irregular gout* makes a general description of this condition impossible. Disorders of the circulatory, respiratory, nervous, digestive and urinary systems, of the skin, bones, joints, and organs of special sense, have all been attributed to goutiness, and often improperly. The only variety about which there can be no question is *retrocedent gout.* Immediately following an acute attack of regular gout, there sometimes occur acute symptoms of gastric, intestinal, cardiac or nervous disorders. They come on suddenly and may subside as suddenly without treatment. The gastric symptoms are those of cardialgia or of acute gastritis; the intestinal symptoms resemble colic with constipation or diarrhea. The cardiac symptoms are those of a mild angina. The nervous symptoms simulate apoplexy or aphasia, or there may be mental aberration.

There is no one pathognomonic symptom of goutiness and the diagnosis must often be made by the family history in the absence of other etiological factors. The diagnosis of the pathological lesion itself may not be difficult, but it is often important to determine the etiological factor before instituting treatment. The affections most frequently associated with the gouty diathesis are catarrhs, neuralgias, muscle cramps, and a form of nephritis called gouty kidney, which presents as its principal symptom occasional uric acid "showers," and deposits of gravel in the urine. Burning and itching of the feet may be due to the gouty diathesis and the chronic eczema of the aged is frequently associated with goutiness. Glaucoma, iritis, keratitis, etc., have been attributed to it. Other diseases, like emphysema, asthma, aneurysms, cardiac inefficiency, thrombosis, hemorrhoids and hepatic congestion, have been ascribed to it. Similarly there have been described gouty pharyngitis, gouty cirrhosis, arthritic colics, gouty phlebitis, gouty myalgia, gouty kidney, etc. Of these the gouty kidney alone gives clear symptoms, pointing to the underlying condition. These are an intermittent albuminuria which may be cyclic, and occasional showers of uric acid, an excess of phosphates or crystals of calcium oxalate.

In many cases of visceral disease the only point in favor of a diagnosis of goutiness is a family history of gout with absence of any other etiological factors. If the disease comes on suddenly, and especially if the urine shows an excess of uric acid,

the diagnosis is strengthened. But even where we are certain that the gouty diathesis exists we must consider all other etiological factors that may produce the condition present and eliminate them before we can make a positive diagnosis of irregular gout.

Treatment.—In most cases of regular gout the physician is first called during an acute attack and then simply for the relief of pain. Colchicum and its preparations have stood the test of time and these are our only trustworthy remedies. But the indiscriminate use of this drug in every case of gout and at every stage betrays an inexcusable ignorance of its action. Colchicum is only of service during an acute attack and such attacks are infrequent in the aged. It is, moreover, a powerful cardiac depressant and gastric irritant and is cumulative in its effects. The dose is 15 minims of the tincture or wine every four hours until the pain is relieved when its use should be stopped. Though usually prescribed in combination with potassium iodide the latter drug has no effect upon the attack, neither shortening nor ameliorating it.

If an acute attack does occur in the aged it is safer to give 1/2-milligram dose of colchicine, repeated if necessary in four hours, but no more for another twenty-four hours. The urine should be made alkaline by the persistent use of potassium bicarbonate and a daily evacuation of the bowels should be secured. For local treatment the application of hot water followed by a cocaine ointment or liniment will afford temporary relief. The pain may be of such severity that it becomes necessary to resort to narcotics. In that case we can give morphine combined with or following a minute dose of atropia. The salicylates and iodoform in grain doses have been given with apparent benefit in some cases. The treatment in the intervals, *i.e.*, between the acute attacks, is mainly hygienic and dietetic. Remarkable results have been obtained at some of the European spring resorts but (as stated in connection with diabetes) it is probable that the benefit derived is due more to the strict regimen than to the effect of the waters, for the same waters taken at home do not produce the same effect, while cases of gout occur even among inhabitants of these resorts who do not follow hygienic and dietetic rules. The principal resorts for gouty patients are Carlsbad, Franzensbad, Marienbad and

Teplitz, all in Bohemia. The hygienic measures are a dry climate, frequent warm bathing, warm clothing to guard against sudden changes of temperature, mild active exercise and a strict regulation of diet. Lime salts and sodium salts, especially common table salt, should be avoided as far as possible. Malt liquors, wines, cider and all fermentation products are to be prohibited. If alcoholic drinks are required we can allow whiskey but no gin. Light meats may be taken but dark meats, liver, kidneys, sweetbreads and other glandular meats are injurious. A vegetable diet is best, but fried dishes, pastries, pies, sweets or candies, and an excessive amount of farinaceous food, tomatoes, rhubarb, and sweet potatoes do harm.

There is no specific treatment for irregular gout. The pathological conditions in the viscera must be treated and the hygienic and dietetic measures given must be adhered to. Among drugs, there is none giving uniform results. Piperazine hastens the elimination of uric acid, and, in some cases, a prolonged course of piperazine water seems to ward off acute attacks; in other cases it is worthless. The same applies to the alkaline treatment with citrate of potash or lithia. The iodides have no effect in this disease. Phosphoric acid has apparently given good results in some cases, but in others it seemed to have had an opposite effect. Diet and hygiene are our only reliable measures.

DIABETES MELLITUS

Diabetes mellitus is a clinical syndrome, the most important symptom of which is a glycosuria, which arises from a defect in the assimilation of carbohydrates, sugar then being found in the blood and tissues which is eliminated by the urine in quantities far exceeding the normal amount.

Glycosuria itself is not pathognomonic of diabetes for it may be produced experimentally by the ingestion of large quantities of sugar. In this case there is no perversion of metabolism, but simply an inability of the normal metabolic processes to completely convert an excessive quantity of sugar at one time. After the excess has been eliminated the urine becomes normal. If the milk glands are removed shortly before or after parturition there is a temporary glycosuria, the process by which glycogen

Paget's Disease Radiogram of lower jaw showing waste of bony structure. (Courtesy of S. Epstein, New York.)

is converted into galactose and milk sugar being halted, and grape sugar, an intermediate product, is then formed. This grape sugar is eliminated by the urine. Glycosuria may also occur during or after diseases of the liver, brain, the ductless glands, infectious fevers, pregnancy and after ingestion of poisons. In some of these cases the glycosuria may persist long after the cause has passed away. Some authorities declare that this form of glycosuria being due to a perversion of carbohydrate metabolism should also be called diabetes mellitus, others will not apply the term diabetes to this temporary glycosuria, as it disappears normally, but apply this term to a more or less permanent glycosuria which in its milder form can be controlled by diminishing the intake of carbohydrates. Still others insist that there is not diabetes mellitus unless the attending symptoms of polyuria, polydipsia, bulimia and emaciation are present. Since neither the nature nor the pathogenesis of diabetes is known, the term will here be applied to any glycosuria that is due to perversion of the metabolism of carbohydrates, whether primary or secondary, temporary or permanent. It should not be applied to simple transitory glycosuria arising from an excessive ingestion of sugar, nor to the glycosuria following amputation of the breast.

Some authors claim that there are many forms of diabetes mellitus depending upon the gravity, stage, probable etiological factor, complications, etc. Diabetes, however, really appears in but two forms, the temporary, self-limited form which is secondary to the diseases just mentioned, and the more or less permanent one, which is really the true diabetes mellitus.

True diabetes mellitus may present its accompanying symptoms in a marked degree or may appear with symptoms so mild as to be unnoticed. In some cases a progressive loss in weight first attracts the attention of the patient, in other cases there may be a vague feeling of malaise without any clearly defined symptoms and an examination of the urine is necessary to clear up the diagnosis.

Notwithstanding an enormous amount of research work in metabolism, the processes by which carbohydrates are converted into glycogen and from glycogen into the various sugars and fats, is still undetermined.

It is believed that glycolitic agents, in the nature of ferments,

exist in the pancreas, kidneys, lungs, white blood corpuscles, etc., and that these ferments cause the transformation of glycogen into sugar. A deficiency or a perversion of the functions of these ferments interferes with the complete combustion of the sugar and it is retained in the blood to be eliminated by the urine. An injection into a dog of diabetic urine, from which the sugar had been removed, will produce glycosuria, and the same result follows if the intestinal contents of a diabetic person are injected into the intestine of a dog. A glycosuria is also produced in animals by the injection of adrenal extract. In these cases lesions of the pancreas are found, evidently due to the action of the adrenalin upon the pancreatic cells of the islands of Langerhans. The adrenals do not themselves affect carbohydrate metabolism. It is their overstimulation that causes the production of excessive secretion which interferes with the nutrition of the pancreatic cells, the function of these cells being, probably, the secretion of the glycolitic ferment. In about 50 per cent. of diabetic cases these cells are found in a state of hyaline or granular degeneration, or in a state similar to that found in other organs undergoing senile involution, *i.e.*, atrophy and sclerosis. The blood in the aged has the tendency to hold the products of incomplete and perverted metabolism, also the products of intestinal decomposition and other toxic matter; likewise an excess of lime salts and waste material, and these abnormal substances do not produce the same constitutional disturbances that appear in younger individuals. For this reason diseases due to disturbed metabolism like gout, diabetes, chronic rheumatism, and some infectious diseases like erysipelas and diphtheria appear in a mild and exceedingly chronic form. As diabetes in the aged is almost always associated with arteriosclerosis, there may be an etiological factor common to both, or the arteriosclerosis may produce malnutrition with consequent degeneration of the cells furnishing the glycolitic ferment.

Many theories have been advanced to explain the production and conversion of sugar, and the causes for the impairment of the chemical processes involved. Since many of the theories apply to some cases or hold good under some circumstances and fail in others, it is evident that there are several causes and various processes that can produce the same end result.

It would serve to no purpose to enumerate these theories or dilate upon the elaborate chemical formulæ used to explain carbohydrate metabolism. Magnus Levy has pointed out sources of error in theories based upon animal experimentation. In these experiments a rapid, radical and serious damage is done to the organism. In human diabetes the decrease of sugar utilization goes on slowly and progressively and the organism partly adapts itself to the new conditions. Moreover, the dog (most frequently used in these experiments) is a carnivorous animal, and there is some difference in the metabolism of carbohydrates. This may account for the rarity of acidosis in canine diabetes.

While in about one-half of all cases lesions of the pancreas are present and in most other cases lesions of the liver or of the nervous system are found, there are some cases presenting no lesion whatever and apparently there is no etiological factor to account for the disease, while in other cases lesions are found but it is impossible to determine any relation between them and the disease. Extirpation of the pancreas is followed by diabetes, but the disease may be present with a healthy pancreas. Extirpation of the thyroid in the dog was followed, in over 60 per cent. of experiments, by diabetes yet diabetes is found complicated by or associated with Basedow's disease.

This much is certain: there are numerous factors which can disturb carbohydrate metabolism, and this disturbance may occur anywhere between the intestines, *i.e.*, the point of ingress into the circulation, and the kidneys, the point of egress. It may result from some lesion in one of the organs producing the glycolitic agent, or it may occur without such lesion, as a result of functional disturbance in cells engaged in the process of metabolism. The sugar is derived from the carbohydrates taken into the system, but it may also be derived from the proteins, Kulz having found that in a diabetic kept on an exclusive protein diet, increase in this diet increased the sugar output, while Pfluger found that the sugar may come from the proteins of the body. It is possible that some sugar is derived from the glycerine of fat.

Etiology.—Statistics show a constantly increasing proportion of diabetics in civilized countries. This may be ascribed to increased mental and nervous strain with decreasing physical

exercise and to changes in the mode of life brought about by the introduction of new methods of preparing food, and of food that is too rich.

Heredity seems to have some influence as an etiological factor, but it is a question whether such influence is really inherent or is simply the result of similar environment and mode of life. The relative frequency of diabetes among Jews is probably due to the fact that they are mostly engaged in sedentary or non-active occupations and their mode of life favors mental and nervous strain. The disease occurs occasionally in families having a gouty diathesis or a disposition to obesity, but while some see therein an argument in favor of heredity, it is probably simply coincidence. Diet is an uncertain etiological factor, some authorities claiming that a vegetarian diet predisposes to diabetes, others showing the comparative rarity of diabetes among peasants who live almost exclusively on a vegetable diet. Diabetes does not occur more frequently among sugar and candy workers than among others and while the consumption of sugar is far greater among females, diabetes occurs in only one-third as many females as males. It is found frequently, however, among obese beer drinkers. In cases where the disease is traceable to faulty alimentation, either in carbohydrate excess or disproportion, there is probably a dyscrasia or predisposition to this disease.

It is often impossible to determine what the exciting cause is. The disease sometimes follows a shock or fright, more often there has been a long period of worry or mental strain. It occasionally follows cerebral traumatism. Many cases follow acute general infectious diseases, and it has been found after local infections. In by far the largest number of cases, however, there is a disease of the pancreas or liver. In other cases there is a nervous or mental defect, a neurosis or psychosis preceding or accompanying the diabetes. Since we do not know the pathogenesis of the disease, we frequently assume a causal relation without any other basis than absence of other etiological factors. The temporary glycosuria of secondary diabetes may produce a permanent diabetes.

Pathology.—In some cases no pathological lesion or condition can be found, except an excess of sugar in the blood, the proportion being as high as 1 to 250 instead of 1 to 1000 or less. Fat granules

may appear in the blood plasma. The most frequent pathological condition is a degeneration of the cells of the islands of Langerhans in the pancreas. There is occasionally a pancreatitis or a degeneration in some other part of the organ. The liver is often hypertrophied, but in senile cases it is generally atrophied and sclerotic. In the rare bronzed diabetes in which the viscera and skin are pigmented there is a pigmentary hypertrophic cirrhosis of the liver, the pigmentation of the other organs being probably secondary to the change in the liver. In the kidneys there is frequent evidence of nephritis. Various other kidney lesions have been noted, but these may have been incidental complications. The same can be said of other lesions occasionally found in diabetic cases, as some are undoubtedly secondary to arteriosclerosis.

Symptoms.—The disease is usually well advanced before any symptoms pointing to it make their appearance. In one case the patient did not notice any loss in weight or strength, or excessive thirst until two years after a glycosuria was accidentally discovered. In the *temporary diabetes* following an infectious or other disease the patient makes a slow recovery and does not regain strength and weight as fast as he should in normal convalescence. The appetite improves, but there is no corresponding gain in weight, and there is a polydipsia, though it is not as marked as in the permanent form of diabetes. The urine is slightly, if at all, increased in amount, but it contains from 1/2 to 2 per cent. of sugar. A diminution in the intake of carbohydrates will diminish the quantity of sugar; however, it is rarely necessary to resort to an exclusive protein diet to get a sugar-free urine. By simply limiting the ingestion of carbohydrates in these cases the glycosuria will disappear.

In the *permanent diabetes* the earliest symptom is usually a loss in strength, often ascribed to ageing. The patient notices that, in spite of a good appetite, he loses in weight, and it is for this loss in weight that he seeks medical advice. Close questioning may then bring out the additional symptoms thirst and polyuria. Ageing patients do not pay attention to these symptoms until they become severe, but will readily notice loss in weight and strength and, ascribing these to age, they become depressed. When the disease is well advanced the mouth becomes dry and the tongue red, glazed and furrowed. The urine is greatly

increased in quantity necessitating frequent micturition. The appetite increases until there is a constant desire for food even after the patient had just finished a hearty meal. This bulimia is greatly aggravated as soon as the carbohydrates are reduced in the course of treatment, and where wheat in the form of bread is withdrawn, the appetite may be insatiable. The thirst keeps pace with the polyuria, which in turn increases as the amount of sugar increases. When associated with arteriosclerosis the symptoms of the latter disease appear in high blood pressure, headache and vertigo, mental and emotional depression. The temperature is often subnormal. Constipation is a frequent complication and is often associated with gastric disturbances. Nervous symptoms appear, especially when the disease is of nervous origin. There may be neuralgia, muscle pain, paralysis, etc. The skin becomes dry and a slight trauma, such as a scratch or the prick of a pin, will often become an extensive and serious lesion. These surface lesions rarely heal without suppuration, and if deep-seated, they may become gangrenous. It is probable that the frequency of furuncles, carbuncles, chronic ulcers and gangrene in diabetic cases is due to the increased amount of sugar in the blood, the blood thereby becoming a good culture medium for the pyogenic cocci. Eczema of the genitals and herpes zoster are occasional complications. The most important symptom, however, is glycosuria. The quantity of urine is generally dependent upon the amount of sugar, although in diabetes following cranial traumatism we may find a polyuria (5 to 6 liters in twenty-four hours) with a sugar content of but 1 1/2 to 2 per cent. Usually, if there is over 5 per cent. of sugar, there will be from 4 to 5 liters of urine in twenty-four hours, and as the sugar percentage sinks the total quantity of urine diminishes. Cases passing as much as 28 liters in one day have been reported. The excretion is generally more voluminous at night than by day and there is often a retention of a few drops which pass away a few moments after the bladder had been apparently emptied. If dropped on the clothes and dried, there will be a deposit of sugar. Naunyn gives the following figures which show the relation between the amount, specific gravity and sugar percentage.

2 liters passed in 24 hours should have a specific gravity of 1028 to 1030 corresponding to 2 to 3 per cent. sugar.

3 liters passed in 24 hours should have a specific gravity of 1028 to 1032 corresponding to 3 to 5 per cent. sugar.
5 liters passed in 24 hours should have a specific gravity of 1030 to 1035 corresponding to 5 to 7 per cent. sugar.
6 to 10 liters passed in 24 hours should have a specific gravity of 1030 to 1042 corresponding to 6 to 10 per cent. sugar.

The amount of urea is generally increased and this is probably due to the increased ingestion of proteins and if much meat has been taken there may be also a considerable amount of uric acid.

Albumin is frequently found in the urine of diabetics. In some cases it is a symptom of a complication, as nephritis, in other cases it is due to the excessive protein food introduced in the course of treatment, in still other cases it is apparently due to a faulty metabolism of proteids, which accompanies the metabolic defect in diabetes. Many theories have been advanced to explain the presence of this albumin, but none are satisfactory. Still more unsatisfactory are the theories advanced for the presence of amino-acids and acetone bodies found in the urine of late cases of diabetes. Acidosis occurs only in grave cases, and rarely in the aged. Phosphaturia is a frequent complication.

Normal blood contains about 1/4 part of sugar in 1000, which may be increased to 1 part or 1 1/4 part immediately after the ingestion of a considerable amount of saccharine matter, but several hours later the sugar proportion has dropped to normal. In diabetes the sugar in the blood may be increased to 3 or 4 parts in 1000, but the proportion varies according to the ingestion of carbohydrates and gravity of the disease. The amount of fat in the blood is generally increased and may reach the proportion of 270 parts in 1000 (Frugoni). In the early part of the disease the blood is hydremic, but after polyuria becomes pronounced it is concentrated, with a specific gravity of 1030 to 1059. The blood is sometimes lighter in color, probably due to the fat, as the hemoglobin content is usually normal.

Sugar is sometimes found in the sweat, occasionally in ascites and other serous transudations, but rarely in the saliva. Diabetic coma, which usually occurs in younger individuals at the closing stage of the disease, is infrequent in old age. This coma is probably due to the toxic effects of the acetone bodies. It may come on slowly or rapidly and death may follow in a few hours or it may be delayed for several days. The diagnosis

of diabetes mellitus is simple, but error may occur if the patient is first seen during the comatose state. If the diagnosis cannot be determined from the history, it may be necessary to withdraw the urine by means of a soft catheter. If there is no sugar it is not diabetes. Diabetes in the aged is generally mild but extremely persistent. Under a restricted carbohydrate diet patients may live for years without discomfort. Carelessness in diet or a sudden shock may increase the sugar output and result in acidosis.

Treatment.—Since we do not know what prevents the complete combustion of sugar in diabetes, the only rational method of treatment is to limit the ingestion of carbohydrates. The dietetic treatment is still our main reliance in the control of this disease, supplemented by measures which have given favorable results in some cases. The only unalterable rule is to diminish the amount of the carbohydrates, but while in some cases it will be necessary to increase the caloric value of the food, in other cases, the patient will do better if the caloric value is not increased, but in the obese, or where the disease is far advanced, it must be diminished. The aged diabetic requires a diet containing about the same caloric value as in health, which is about 30 calories per kilogram weight daily, or about 2000 calories at a weight of 145 pounds.

It is rarely necessary, nor is it advisable, to make a sudden and profound change in the diet by excluding carbohydrates entirely, as in all early cases, and in many advanced cases too, the organism can tolerate a certain amount of carbohydrate food without the production of sugar. It is, therefore, necessary to determine the point of carbohydrate tolerance and this can be done by placing the patient upon Von Noorden's Standard Test Diet which is as follows:

Von Noorden's Standard Test Diet

Breakfast.—200 grams coffee or tea with one or two tablespoonfuls of thick cream.

100 grams of hot or cold meat (weighed after cooking).

Two eggs, with or without bacon or corned beef.

50 grams of white bread.

Lunch.—Two eggs cooked as desired, but without flour; or any other hors d'oeuvre free from flour.

Meat (boiled or roasted), fish, venison or fowl, according to taste, about 200 to 250 grams altogether (weighed when cooked).

Vegetables, such as spinach, cabbage, cauliflower or asparagus, prepared with broth, butter or other fat, eggs or thick sour cream, but without any flour.

20 to 25 grams creamy cheese (such as Brie, Camembert, etc.); plenty of butter.

Two glasses of light white or red wine, if desired.

One small cup of coffee with one or two tablespoonfuls of thick cream.

50 grams of white bread.

Dinner.—Clear meat soup, with egg or green vegetable in it.

One or two meat dishes as at lunch.

Salad of lettuce, cucumber or tomatoes.

Wine.

No bread.

Drinks during the day, exclusive of wine, one or two bottles of aerated water.

The total urine excreted during the twenty-four hours is collected, that of the day and of the night separately, and is examined quantitatively for sugar. Both the percentage contents, and more especially the whole quantity of sugar excreted in the twenty-four hours is noted.

If on this fare no sugar is excreted, the quantity of bread is gradually increased until sugar does appear in the urine. If on the other hand, sugar is excreted with this test diet, the patient is first kept on the same fare until the daily excretion of sugar has become nearly constant. Then the quantity of bread is gradually diminished. At each stage in the diminishing process the patient is kept on the same amount of bread long enough to allow the sugar excretion to get a constant value, proper to this stage.

The largest amount of white bread which can be taken without causing sugar to appear in the urine is then taken as that particular patient's point of carbohydrate tolerance.

These meals allow 100 grams of white bread having a 60 per cent. starch content daily. When the point of carbohy-

drate tolerance has been determined the amount of white bread can be replaced by other carbohydrates according to the following table of carbohydrate equivalents:

TABLE OF EQUIVALENTS

30 grams of white bread equal in carbohydrate contents

Breads and Other Farinaceous Foods

Brown bread	40 grams
Corn bread	40 grams
Rye bread	36 grams
Graham bread	36 grams
Gluten bread	36 grams
Biscuit	32 grams
Roll (French)	32 grams
Roll (Vienna)	32 grams
Crackers (Boston)	24 grams
Crackers (Graham)	24 grams
Crackers (Oyster)	24 grams
Pretzel	24 grams
Ginger bread	28 grams
Chocolate cake	28 grams
Sponge cake	28 grams
Cookies (molasses)	24 grams
Lady fingers	24 grams
Doughnuts	32 grams
Spaghetti	120 grams
Macaroni	120 grams
Vermicelli	120 grams
Almond meal	260 grams
Soja bean meal	200 grams
Potato gluten biscuit	180 grams
Pure gluten biscuit	200 grams
Barkers gluten food, A	409 grams
Barkers gluten food, B	296 grams
Barkers gluten food, C	216 grams
Vegetable gluten	68 grams
Gum gluten	48 grams
Glutona	32 grams
Glutosac bread	60 grams
Protopuff No. 1	180 grams
Protopuff No. 2	48 grams
Jireh whole wheat bread	48 grams

Vegetables

Celery	150 grams
Radishes	450 grams
Asparagus	550 grams
Cabbage	310 grams
Cauliflower	400 grams

TABLE OF EQUIVALENTS.—(*Continued*)

Cucumber	600 grams
Lettuce	600 grams
Mushrooms	250 grams
Sauerkraut	450 grams
Spinach	600 grams
Tomatoes	450 grams
Beets (cooked)	260 grams
Lima beans	200 grams
Carrots	260 grams
Corn (canned or green)	88 grams
Egg plant	360 grams
Parsnips	140 grams
Green peas	120 grams
Potatoes	88 grams
Sweet potatoes	30 grams
Turnips	224 grams

Cereals

Barley (cooked)	27 grams
Hominy (cooked)	100 grams
Oatmeal (cooked)	160 grams
Rice (cooked)	60 grams
Farina (cooked)	100 grams

Fruits

Apples	180 grams
Bananas	80 grams
Grapes	128 grams
Muskmelon	448 grams
Oranges	160 grams
Peaches	200 grams
Pears	200 grams
Prunes	96 grams
Strawberries	260 grams
Watermelon	900 grams
Cherries	150 grams
Blackberries	160 grams
Cranberries	180 grams
Currants	160 grams
Raspberries	150 grams
Grapefruit (weighed with skin)	750 grams

Desserts

Apple pie	40 grams
Lemon pie	36 grams
Custard pie	78 grams
Rice pudding	56 grams
Tapioca pudding	60 grams

TABLE OF EQUIVALENTS.—(*Continued*)

Milk and Milk Products

Pure milk	448 grams
Cream	448 grams
Koumyss	334 grams
Matzoon	886 grams
Kefir	750 grams
Buttermilk	375 grams
Condensed milk (sweetened)	33 grams
Condensed milk (unsweetened)	144 grams
Evaported cream	144 grams

Beverages

Beer (dark)	250 grams
Beer (light)	300 grams
Ale	298 grams
Porter	238 grams
Sherry wine	510 grams
Port wine	258 grams
Champagnes	108 grams
Rhine wines (red)	570 grams
Rhine wines (white)	600 grams
Italian wines	495 grams

Miscellaneous

Cocoa (unsweetened)	50 grams
Chocolate (unsweetened)	60 grams
Peanuts	80 grams

If for example the point of carbohydrate tolerance is found when 60 grams of white bread are taken in a day, 30 grams of the bread can be replaced by 120 grams of spaghetti or by 100 grams of lima beans, 112 grams of cream and 112 grams of muskmelon. If bread is entirely omitted the craving for this one article of food becomes intolerable and the patient will either starve or violate orders. The ordinary gluten bread contains almost as much carbohydrate as the white bread and most of the so-called diabetic gluten breads contain a large percentage of starch. The only diabetic flour containing no starch is casoid flour, which is a mixture of albuminoids. If this is substituted for the ordinary white bread a much larger quantity of other carbohydrates can be taken. Diabetics differ in their tolerance toward certain foods, the same carbohydrate equivalent of one food producing a glycosuria in one and not in another who has the same point of tolerance. Articles toward which there is an intolerance must

be avoided. Foods that are absorbed slowly, such as contain a large amount of cellulose for example, are better than those that are rapidly absorbed. The oatmeal cure recommended by Von Noorden consists of the daily administration of from 200 to 250 grams of oatmeal, preferably in the form of gruel in divided doses at intervals of two hours. In addition to this, from 200 to 300 grams of butter and 100 grams of proteid food are allowed. Black coffee or tea, good old wine or a little brandy is permitted. After three or four days of this diet the patient is placed upon a vegetable diet for a day or two, the vegetable content not to exceed the point of carbohydrate tolerance. Various theories have been advanced to explain the frequent success in diminishing the quantity of sugar and acetone under the oatmeal diet. It has been suggested that the large amount of cellulose in oatmeals causes very slow absorption, or that the large amount of water in gruel diminishes the total quantity of the oatmeal, again that upon a single carbohydrate diet the appetite wanes and less of all kinds of food is taken, or that the oatmeal is converted beyond the stage of sugar. Some of these explanations apply as well to other single carbohydrate diets, as for example to the potato cure, rice cure, etc. It is certain that the glycosuria is diminished whenever the diet is restricted for a few days to a single carbohydrate and smaller amounts of protein and fat than normal. If excessive sugar persists in the urine, notwithstanding the diminution of carbohydrates to the point of exclusion, the intake of protein must be reduced. These cases, however, are rare in the aged. In many instances it is possible to reduce the amount of sugar in the urine to 1 or 1 1/2 per cent. without great restriction of carbohydrates, and only complete exclusion of starch and sugar from the diet will bring it down to normal. If the patient feels well with a glycosuria containing 1 or 1 1/2 per cent. of sugar and does not lose weight, the point of carbohydrate tolerance should be established upon that basis. The indiscriminate use of fats in diabetes may lead to acidosis. Stern has shown that the fats containing a large proportion of fatty acids of a low molecular weight favor the production of acetone, but, if the fatty acids have a high molecular weight, they yield little acetone. This would exclude from the diet butter and cream, but not olive oil, lard or suet. He recommends the yolk of eggs as the most valuable fatty substance in diabetes,

especially in acidosis. Instead of sugar, saccharine or levulose can be used to sweeten coffee and tea and when these become distasteful glycerin may be used instead. It is impossible to arrange a strict diet list for the aged diabetic, because the disease is rarely severe in them, therefore much greater leeway can be permitted in the matter of diet to maintain physical strength. An exclusive protein diet would produce gastric and intestinal disturbances and would so far reduce the patient's strength that recovery would be impossible.

Non-dietetic measures include aerotherapy, electrotherapy, hydrotherapy, hygienic measures, surgical measures and drugs. Abrams in his work on spondylotherapy recommends concussion of the seventh cervical vertebral spine and reports cases where diabetes has been benefited by this method of treatment. It is well known that diabetes in hot countries is milder and more prolonged than in colder countries and this has led to the dry-heat treatment. In a dry air with a temperature of from 80 to 90° F. the glycosuria diminishes and the symptoms of diabetes become milder. Upon exposure to cold the glycosuria and other symptoms become as pronounced as before.

The treatment by electricity has not been satisfactory. DeKraft reports cures from the employment of high-frequency currents, Tousey thinks they may be harmful, Stern says they do not influence the intensity of glycosuria, azoturia or acetonuria. Other observers make similar contradictory reports.

Many cases of diabetes are apparently cured at the Bohemian mineral springs, especially at Carlsbad and Franzensbad. It is hardly possible that the waters themselves effect the cure, since the waters taken at home do not produce the same results. This was shown in the case of a man, age sixty-five, with symptoms of diabetes and a sugar content of 5 per cent., who after a six weeks' course at Carlsbad gave a sugar content of but 1 1/2 per cent. It rose soon after his return, however, and in two months it again reached 5 per cent., notwithstanding a partly restricted diet. The following year the same course was followed by the same result. The third year he took the waters at home, following, to a modified extent, the strict routine and diabetic regimen insisted upon at Carlsbad, and his sugar content dropped to 2 1/2 per cent. The next year he returned to Carlsbad and there was again the usual result, diminished glycosuria and

relief of other symptoms. Undoubtedly the psychic influence of the environment and strict regimen were the most important factors. Free intestinal elimination is an important adjunct to the dietetic measures, and in some cases free catharsis with restricted diet may effect a cure. Any of the saline cathartics act equally well.

Medicinal measures are usually required to relieve symptoms or to prevent complications, and sometimes they are employed as a general tonic. Occasionally medicinal remedies are given to cure the disease and there are reports of recoveries from the use of some drugs. In every case, however, the dietetic measures must be included in the treatment. The use of uranium nitrate in 5-grain doses will sometimes reduce the amount of sugar. Methylene blue, strontium lactate, chloride of gold and sodium, iodoform, antipyrin, mercury bichloride, and arsenic have all been recommended, yet they almost invariably fail to give the results obtained by those who advocate their use. Sewall reports an absence of sugar after the administration of an infusion of lean meat acidulated with hydrochloric acid and Horowitz reports a like result from the administration of the lactic acid bacilli. Rudisch recommends atropine sulphate in doses of from 1/150 grain gradually increased to 1/20 grain and atropine methylbromide in doses of 2/15 grain gradually increased to 8/15 grain, while Stern obtained only toxic effects from these large doses. Codeine has stood the test of years and is still frequently used when dietetic measures alone fail to reduce the quantity of urine.

It is, however, rarely necessary to resort to other than dietary measures in senile cases except for the relief of distressing symptoms and as a prophylactic to prevent coma. When acidosis appears sodium bicarbonate or potassium bicarbonate must be given in doses of 10 grains repeated every 4 hours. If coma supervenes in spite of the alkaline treatment, it almost invariably ends in death. A few recoveries are recorded, however, following the intravenous injection of soda bicarbonate, using 500 c.c. of a 3 per cent. solution, and giving one or two injections daily. Large quantities of the salt may be required and Hanssen reports a case in which 240 grams were given in 10.6 liters of water in ten half hourly doses. For the relief of thirst the valerianates, ammonium valerianate or quinine valerianate in 5-grain doses,

can be given. Water acidulated with phosphoric or citric acid (sweetened with glycerin) and small pieces of ice are of temporary utility. Bulimia can be temporarily controlled by cocaine hydrochlorate given in 1/8-grain doses. The cocaine, however, is a cardiac depressant, and if frequently repeated, will cause habituation and gastric atonicity. Food containing a large amount of cellulose, or requiring much chewing, as underdone meats, should be taken in small quantities and eaten slowly.

Headache is usually due either to acidosis or arteriosclerosis and the treatment depends upon the cause.

Jaundice is due either to pancreatic or hepatic disease. If it disappears under the administration of calomel given in repeated small doses, say 1/10 grain every two hours, or sodium glycocholate in 2-grain doses twice daily, we have a diabetes of hepatic origin to deal with. A persistent jaundice under this treatment does not exclude disease of the liver, but it points with greater force to pancreatic diabetes. Digestive and intestinal disorders are frequent complications of diabetes, due, no doubt, to the changed alimentation and changed character of the blood, whereby the nutrition of the organs becomes impaired. Occasional lavage will increase the activity of the stomach and remove food particles that have begun to decompose. For constipation the most effective treatment is a pill containing dried ox gall 5 grains and aloin 1/4 grain, at night, followed in the morning by a saline. Diarrhea indicates a catarrhal condition of the bowel generally due, in the aged, to excessive food, occasionally to intestinal fermentation and irritation. In the latter case the stools are foul-smelling. This condition can be relieved by intestinal antiseptics. Fatty diarrhea and steatorrhea indicate involvement of the pancreas or liver or perhaps of both. Beside the treatment of the causative condition the fat ingesta must be diminished.

Albuminuria is found in about 40 per cent. of all cases. A trace of albumin is generally present in the urine of the aged and signifies a senile contracted kidney. Unless there are symptoms of nephritis (casts, etc.), or when albuminuria appears suddenly in a large amount, it may be disregarded.

Loss of virility is a frequent accompaniment of diabetes. This is probably due in most cases to the male climacteric

which occurs about the end of the fifth decade and to the natural loss of virility due to ageing. It has no significance apart from the mental depression that its discovery occasionally produces. Nothing should be done for it.

Ocular and aural disorders sometimes complicate matters. Diabetic cataract may improve when there is an improvement in the general disease but other disorders require special treatment directed to the organ involved.

Pruritus is a frequent accompaniment and occasionally it is the most annoying symptom. Its favorite sites are about the genitals and anus where it is often associated with bromidrosis and intertrigo and about the legs. In moist locations the treatment must be directed to the hyperhidrosis. Stearate of zinc and salicylic acid will generally relieve the excessive secretion and, if the itching continues, inunction with a 2 per cent. cocaine ointment, using lard or any animal fat as a base, will afford temporary relief. This ointment can also be used in localities where the skin is excessively dry. The intensity of the pruritus depends upon the intensity of the glycosuria. The resistance of the diabetic to infectious disease, especially to septic infection, is considerably lowered, the opsonic index being approximately one-third lower than normal. It is probable too that the sugar-laden blood is a better culture medium than normal blood. We find consequently streptococcus and staphylococcus infections (in the form of furuncles, carbuncles, abscess, cellulitis and septic gangrene) frequently attacking diabetics. Dry gangrene occurring in diabetes is not due to that disease but to arteriosclerosis, embolus or other cause that prevents nutrition of the part; the greater tendency to infection may, however, convert a dry gangrene into a purulent one. Serum therapy in the form of a vaccine injecting the three varieties of the staphylococcus pyogenes, aureus, albus and citreus, has been found curative in some cases of diabetes with infection when used at the onset of the complicating lesion. If this fails, local treatment must be instituted. Furuncles generally, and occasionally carbuncles at their onset, can be aborted if a needle at a white heat is thrust into the center of the inflamed elevation. In more advanced cases of carbuncle, cauterization by lunar caustic or caustic potash, or else excision, is necessary. Carbuncle in the aged is a grave disease and com-

plete excision at the earliest possible moment is in most cases the only method of successfully dealing with it.

The treatment of gangrene in diabetes is purely surgical. The serum therapy may be tried, but unless there is rapid and marked improvement no time should be lost in local medication. The danger from operative procedure is less than the danger from septic gangrene, and with modern methods of anesthesia and surgery, operations upon diabetics are no longer prejudged fatal.

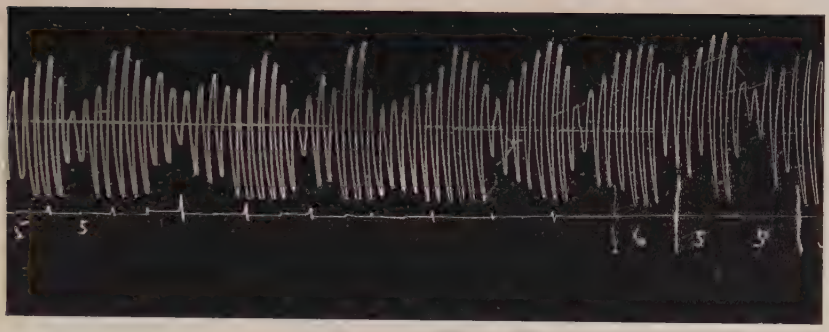
CEREBRAL HYPEREMIA

Cerebral hyperemia does not differ in the aged from the same condition in maturity. Passive congestion, however, is more frequent, being often due to the impaired jugular circulation following dilatation and tricuspid disease.

Active hyperemia in the senile occurs after excitement, physical exercise, excessive food, coffee, tea or alcohol. It begins with a sense of heat in the head, then a fulness and a beating with throbbing temples, spots before the eyes, buzzing in the ears, vertigo and a dulling of the intellect tending to unconsciousness. The face is flushed and conjunctivæ are injected, carotids are prominent and their impulse is marked. The attack passes away in a few minutes if the cause is removed.

Passive hyperemia is a chronic condition due to venous stasis. It may occur as a temporary condition if the cause is of a temporary nature, as the pressure of a tight collar, stooping, coughing, etc. In the permanent condition there is either a valvular disease which interferes with the return circulation, or else some local interference, such as a growth pressing upon the jugular. The symptoms are persistent headache, drowsiness with inability to sleep when lying down, vertigo, flushed face and marked prominence of the jugulars. The arteries, however, are not prominent.

Treatment of cerebral hyperemia depends upon the form and the cause, which must be removed whenever possible. This can generally be done in active hyperemia and in those cases of passive hyperemia or venous stasis, the causes of which are temporary, as external pressure. In cases due to interference with the venous circulation, following cardiac disease, or the pressure



Tremorgraph—Paralysis agitans, left hand. (Neustaedter, *Med. Record*, July 17, 1909.)

Early paralysis agitans; facial aspect characteristic. Attitude not yet pronounced. (Dr. M. Neustaedter's case.)

of a tumor, we must resort to temporary measures to lessen the cerebral circulation. The most effective measures for this purpose are hot foot or sitz baths. Local hyperemia in other parts of the body may be produced by mustard, turpentine stupes, hot cloths, cold to the head, etc. If there is danger of apoplexy, leeches to the temples or cups to the chest and back, or venesection must be employed. Drugs are useless. Ergot which is serviceable in earlier life is dangerous in the aged if there is cerebral arteriosclerosis. The opiates and alcohol increase the hyperemia and chloral is dangerous on account of its depressing action upon the heart. The bromides will relieve reflex irritation and veronal can be used for the insomnia. The head must be kept raised and the feet lowered even in sleep. Rapid catharsis will sometimes relieve the hyperemia if due to excessive food or coffee or alcohol. The coal-tar preparations acetphenetidin, acetanilid and similar preparations depress the heart and leave it depressed.

PARALYSIS AGITANS

Paralysis agitans is a progressive motor neurosis of middle and advanced age. Neither its cause nor its pathology are known, there is no lesion distinctive of the disease, and of the many theories advanced for its pathogenesis, none is satisfactory.

Etiology.—The basic etiological factor is unknown. At the present moment, we ascribe the cause for anatomical and functional perversions to microorganisms or to perverted internal secretions. It has been suggested that paralysis agitans may be due to atrophy of the parathyroid glands, but it is more probably due to a senile change in the motor branches of the spinal nerves, although no change in them has been demonstrated. There is generally a neurotic tendency, and in many cases an etiological factor affecting the brain or spinal cord can be discovered. It may be shock, fright, intense emotion, prolonged worry or fear, or overwork. In some cases there is a history of traumatism, in others an acute infectious disease preceded the advent of the paralysis agitans. Exposure to cold, dampness, unhygienic surroundings and poverty have been given as the exciting causes, while in many cases no cause can be found.

Pathology.—The only pathological condition present in every

case is an arteriosclerosis of branches of the spinal arteries. This has, however, been found in cases which did not present the symptoms of the disease and on the other hand far advanced paralysis agitans presented on autopsy but slight vascular changes.

Symptoms.—Paralysis agitans presents a characteristic clinical picture. Though a tremor disease, there are cases without tremor, cases where the tremor is temporarily absent, but the attitude and gait are always present. When standing the patient is bent over as though he were about to fall forward, his knees and elbows are bent, the hands are held in the position of holding a pen or rolling a pill. The walk corresponds with the attitude, the bent position being maintained, and there is a forward pitch with short, rapid, shuffling steps which must be kept up until an obstruction is met with. In the early stage of the disease the patient can stop himself, but when the disease is well advanced he will continue to go forward until stopped or until he falls and if pushed or pulled backward he will continue going backward until stopped. The attitude is due partly to a gradually increasing rigidity of the muscles first of the neck and back, later of the extremities and face. As a result of this muscle stiffness, voluntary movements become difficult, slow and deliberate. This is well seen in the hand writing which, aside from the tremulousness, becomes so small and cramped as to be almost illegible. Owing to the rigidity of the muscles of the face it becomes expressionless, not apathetic as in dementia, but mask-like.

The tremor, which in most cases is the earliest and most pronounced symptom, usually begins as a fine trembling in one hand, then the leg of the same side is affected, later the opposite leg and lastly the other hand. The order is not regular and in some cases the tremor is confined to the hands or feet alone, or to the extremities on one side. Late in the disease the head and neck muscles are affected and there is a coarse shaking or nodding of the head with tremor of the lower jaw and lips. The tremor rate is from four to seven per second but it may be temporarily controlled by the will and it ceases during sleep. Excitement and fatigue do not increase the rate but they increase the extent of the oscillations until they become a coarse tremor or shake.

"Paralysis agitans sine tremore," paralysis agitans without

tremor, is occasionally met with. The name is a misnomer, for while there is progressive weakness there is never complete loss of power, and without tremor there is no agitation.

In these cases there is progressive muscle rigidity, generally marked on one side and but slight on the other, and later the characteristic attitude appears. A peculiarity of the gait is an apparent inability or lack of energy to make the first step when intending to walk. It then requires some powerful mental impression as a threat, or some external impetus, to start him.

Diagnosis.—When the disease has so far advanced that the characteristic attitude becomes a prominent feature error in diagnosis is impossible.

Before this time it may be mistaken for senile tremor (see Senile Tremor) and other tremor diseases. Multiple sclerosis and hysteria are extremely rare in old age. The different character of the tremor and the absence of muscle stiffness will exclude these and also the toxic tremors (lead, mercury, alcohol, etc.).

Treatment.—The disease is incurable. It progresses slowly and while cases have succumbed within a year after the initial symptoms were observed, the ordinary duration is from five to fifteen years.

Hyoscine in 1/300-grain, or hyoscyamine sulphate in 1/100-grain, doses, hypodermically, will relieve the tremor and muscle stiffness, but the dose must be constantly increased and the drug finally discontinued when toxic effects appear. Duboisin may then be substituted and given in doses of 1/150 grain, gradually increased. Hydrotherapy and electrotherapy are of temporary utility in arresting the progress of the disease. Arsenic has a more permanent effect and should be given until the physiological effects of the drug compel its discontinuence. Nux vomica which has been recommended as a tonic seems to intensify the tremor and muscle cramps. When the patient becomes bedridden measures must be employed to prevent hypostatic congestion and edema.

PROGRESSIVE BULBAR PARALYSIS

Glossopharyngolabial paralysis is a rare disease, occurring most frequently in advanced life. It is a symmetrical paralysis affecting the muscles of the lips, tongue, palate, pharynx, larynx and the muscles of mastication.

Etiology.—The cause is unknown. Supposed causes are shock, strong emotions, cold, overexertion of the muscles supplied by the hypoglossal and glossopharyngeal nerves, injury and infections.

Pathology.—There is a degeneration of the motor nuclei of the medulla and pons, the ganglionic cells atrophy, the hypoglossal, glossopharyngeal, facial, vagus, accessory and sometimes trifacial motor trunks are degenerated and the pyramidal tracts of the cord are occasionally involved. In some cases the anterior horns are atrophied and the lateral columns are degenerated, and we find the same lesions as observed in progressive muscular atrophy and amyotrophic lateral sclerosis, but the symptoms of these diseases are absent or appear late.

Symptoms.—The symptoms are a progressively increasing difficulty in speech, phonation, chewing and swallowing, later difficulty in respiration, a fibrillary tremor of the tongue and muscles of mastication and waste of the muscles involved. The speech becomes difficult, due to gradual paralysis of the tongue, and the linguals are slurred over. Later the same difficulty arises with the labials and the speech becomes indistinct, obscure and finally incomprehensible. At the same time progressive paralysis of the larynx and vocal cords makes phonation difficult and the voice becomes weak, finally dropping to a monotonous hoarse whisper. The tongue slowly loses its motility and becomes completely paralyzed. Owing to the paralysis of the lips, the mouth cannot be closed and saliva drivels out. The patient is unable to whistle. The soft palate drops, further interfering with speech and deglutition. Weakening of the muscles of mastication makes it impossible to move the jaws from side to side and the paralysis of the muscles of deglutition prevents swallowing, the food remaining as a bolus in the posterior part of the mouth while fluids run back through the nostrils. When the disease is fully developed the lower half of the face is expressionless, the lower lip and corners of the mouth hang down and the countenance presents a very peculiar appearance, the lower part being paralyzed while the upper part is active. The mind is unimpaired. There is usually tachycardia, with dyspnea and with exaggerated facial reflexes.

Progressive bulbar paralysis is distinguished from hemorrhage, thrombosis, and embolism in the medulla, by its slow

advent, the other conditions appearing suddenly. Tumors in the medulla produce more extensive paralysis beside intense headaches.

Treatment.—There is no known treatment, death usually occurring in a few years from asthenia. It may occur earlier, however, from aspiration pneumonia, respiratory paralysis or other complicating disease. Strychnine, arsenic, iodides, nitrate of silver, the galvanic and faradic currents have all been used with apparent momentary improvement, but no cure has ever been reported.

ACUTE BULBAR PARALYSIS

This is a form of apoplexy which resembles the progressive bulbar paralysis in its clinical picture but differs from the latter in its sudden onset. It occurs as the result of a hemorrhage into the pons by breaking down of the tissue after thrombosis or embolism in the basilar or vertebral artery, or in one of their branches had taken place. Thrombosis produces premonitory symptoms such as vertigo, tinnitus, headache, insomnia, etc. The onset of the disease is abrupt. A momentary vertigo and vomiting is followed by convulsions. When these pass away there remains a glossopharyngolabial paralysis with all the symptoms described under progressive bulbar paralysis, and frequently also a hemiplegia or paraplegia. The paralysis of the facial muscles, and muscles of deglutition, mastication and speech, is not always symmetrical, and where there has been an extensive extravasation of blood the muscles of the upper part of the face, including the motor muscles of the eyes, may become involved. The disease, when extensive, is rapidly fatal; in mild cases recovery is possible.

Treatment as for apoplexy.

PSEUDOBULBAR PARALYSIS

In this disease the symptoms of bulbar paralysis set in after several apoplectic attacks during which other forms of paralysis had occurred. The ordinary lesions of cerebral apoplexy, cerebral hemorrhages, are found, and in most cases there are minute extravasations in the pons and medulla. The disease

when fully developed presents the symptoms of cerebral arteriosclerosis, cerebral apoplexy and progressive bulbar paralysis and is really a combination of the three diseases. The fifth group takes in diseases which do not fit in any of the preceding groups.

DISEASES UNINFLUENCED BY AGE OR RARE IN OLD AGE

INFECTIOUS DISEASES

The resistance to bacterial influences is apparently greater in old age than in earlier life. This is opposed to the general view that resistance is lowered in the aged, but the simple fact that infectious diseases rarely attack old persons even in epidemics, would seem to substantiate this statement. Whether this resistance is due to an increase in opsonins, more active phagocytosis or a change in the body tissues whereby the tissues become an unsuitable field for the propagation of the germs, is unknown. Other possible explanations are an inherent resistance in senile cells, a lower body temperature, less exposure, etc. The only diseases of this character that are relatively as frequent in the aged as in maturity are erysipelas, variola, influenza, typhus and cholera. The frequency of erysipelas on the lower limbs is readily accounted for by the presence of surface lesions, such as excoriations, scratches, eczema and ulcers in that locality. The frequency of influenza is due to the prevalence of chronic bronchitis, the impaired mucous membrane being a suitable field for the growth of the pathogenic bacillus. That variola frequently attacks the aged in epidemics is probably due to the fact that the immunity secured by vaccination in childhood wears off in the course of years and the aged are hence more susceptible than younger persons who have been more recently vaccinated. The greater susceptibility of the old to typhus is probably due to the general debility which lessens resistance to this disease and the same cause with the senile changes in the intestines will account for the relative frequency of cholera in the aged. Cholera and typhus are infrequent in this country, however, and for this reason cases among the aged here are of the greatest rarity.

The acute infectious diseases when occurring in the aged present some peculiarities. They do not run a typical course nor present a typical temperature curve, and the temperature is rarely as high as in maturity. In the eruptive diseases the eruption is milder and may be absent, but constitutional symptoms are usually graver and death frequently ensues in cases which are apparently mild. In the graver diseases like typhus, cholera and variola, death usually occurs within a few days after the onset of the disease. In the chronic infectious diseases and in those that do not run a definite course the symptoms are usually milder but more persistent than in earlier life.

It is assumed that the physician is familiar with the ordinary etiological factors, symptoms, signs and therapeutic measures employed in infectious diseases in maturity. Where differences in these factors in maturity and senility exist, they will be described, but otherwise the etiology, symptoms, signs and treatment will be omitted. Such rare diseases as malta fever, miliary fever, etc., and diseases which are not known to occur in senility such as rotheln, varicella, etc., will receive no further consideration. In the differential diagnosis between diseases giving similar symptoms, only pathognomonic symptoms and signs where such exist, or else a few cardinal symptomatic differences will be noted. As a rule the temperature range of acute infectious diseases is lower in the aged, the eruption in the exanthemata is lighter, more scattered and the spots fewer in number. They come on later, and whereas they appear in successive crops in maturity, in old age there is only a single crop. The cerebral and nervous symptoms are usually much more pronounced than in earlier life, the prostration is more severe, complications are more frequent, and the grave diseases, like typhoid and variola, are more fatal. In diseases in which the bronchial mucous membrane is seriously involved, bronchial and pulmonary complications frequently end in death. Many cases are followed by incomplete recovery, mental and physical impairment, and foci susceptible to later diseases are retained.

Scarlatina has been reported in the aged. It appears in a mild form, however, and the temperature is but slightly elevated; the eruption is light and the complications of early life are usually absent or are mild. The pharynx and tonsils may be reddened, but there is never the scarlatinal diphtheria nor any cervical

gland involvement, and rarely the typical strawberry tongue. Desquamation sets in early, usually before the end of the first week. Cases of *scarlatina sine exanthemate* also occur in the aged. These cases present the buccal and pharyngeal lesions with fever but not the rash. Desquamation occurs as in ordinary scarlatina. The prophylactic treatment is as in childhood. The only treatment during the disease is rest, the reduction of temperature, if high, by means of warm baths, maintenance of the strength of the heart and of the organism as a whole, and hygienic and dietetic measures. As a mouth wash and gargle nothing equals a solution of peroxide of hydrogen.

Measles occur rarely in the aged and do not differ from the same disease in earlier life, though they appear in a much milder form, the conjunctival symptoms are milder, but the irritation of the bronchial mucous membrane is more severe and may lead to a bronchopneumonia. This is the only danger in measles but it is ever present until the disease has entirely disappeared, and it forms the basis of the usually unfavorable prognosis. The disease itself is mild, the rash is slight, with but little elevation of temperature, but there is a profound feeling of malaise. The symptoms diminish in severity after the first day and may disappear entirely within two or three days. If there is an old bronchitis present, a capillary bronchitis by extension is almost unavoidable. Cardiac disease or arteriosclerosis complicating measles increases the asthenia and makes the prognosis more unfavorable. The treatment is hygienic and symptomatic. If there is conjunctivitis the room should be darkened. For the irritating cough with which the disease usually begins codein, heroin or dionin should be given and menthol and eucalyptol should be inhaled. The disease being usually of short duration, tonics and cardiac stimulants are not required. The danger of extension of the bronchitis to the vesicles can be lessened by the inhalation of steam through an inhaler.

Diphtheria may occur in the aged and is then usually so mild in its subjective symptoms that it may pass unnoticed. There may be little or no elevation of temperature, no pain, swelling or redness of the tonsil, nor any other symptom than the exudate. The exudate does not differ from the diphtheritic exudate of early life, but it is more tenacious and may persist for weeks,

in spite of the use of antitoxin. The mildness of the symptoms is, however, a source of danger as its presence in the fauces may be overlooked until the disease has advanced to the larynx. Laryngeal diphtheria, which is almost always fatal, begins with hoarseness, cough and a feeling of irritation in the larynx as though there were a bit of tenaceous mucus which the patient is unable to bring up. The exudate increases downward over the trachea, causing spasmodic contractions, dyspnea, cyanosis and death. In some cases the cough loosens a fragment of exudate which drops into the bronchus, blocking a tube and causing sudden dyspnea and suffocation. Death is usually not due to the virulence of the diphtheria bacillus but to the local obstruction, or to exhaustion from coughing. In gangrenous diphtheria a gangrenous ulcer appears upon the tonsil, sometimes within a day or two after the initial malaise, the opposite side is rapidly involved and the adjoining tissue in the pharynx, uvula and soft palate may also become gangrenous. There is an abundant secretion of foul-smelling pus, sometimes mixed with blood, grave constitutional symptoms appear, such as extreme exhaustion, weak, irregular pulse, shallow breathing, headache and albuminous urine, containing casts, etc. There is rarely any fever or involvement of the cervical glands. The disease is almost always rapidly fatal. The diagnosis of diphtheria is simple and depends upon the presence of the pathogenic bacillus. The usual antitoxin treatment applies to the aged exactly the same as to younger individuals, but it is never necessary to exceed 3000 units, given in a single dose except in laryngeal cases. Larger or repeated doses will not hasten the removal of the exudate which may persist for weeks, while a single dose of 1000 units may suffice to prevent laryngeal involvement. Diphtheria antitoxin is useless in gangrenous diphtheria, the condition being due to a mixed infection in which a virulent strain of streptococci or staphylococci is responsible for the gangrene. A mixed or autogenous vaccine is required in these cases. In laryngeal diphtheria antitoxin should be used in doses commensurate with the severity of the symptoms.

For the removal of the membrane a 10 per cent. solution of papayotin or trypsin will give the most rapid result. It should be swabbed over the patch every hour or two. The membrane will re-form but the frequent application will prevent the exten-

sion of the growth to the larynx. A solution of peroxide of hydrogen can be employed locally, but if either of the enzymes is employed the peroxide of hydrogen must be used afterward, never before the other. Lactic acid will also remove membrane. Chlorate of potash is useless in diphtheria. The hygienic and dietetic regulations need no special consideration, except that food should not be taken hot, and precautions should be taken to prevent the spread of the disease. Internal medication is not required except where complicating symptoms appear.

Whooping cough differs in some minor factors from the disease in infancy. After a primary catarrhal period lasting from two to three weeks, during which there is a bronchial catarrh, constantly increasing in intensity, the second period sets in with a convulsive cough. The paroxysms of coughing are similar to those of infancy, but they occur more frequently during meals, the movements of deglutition apparently provoking the attack. During the cough of the aged, the characteristic whistling inspiration observed in infants is absent and vomiting which is a frequent accompaniment of the cough in the young does not occur in the old. After an indefinite period the spasmodic attacks cease and a bronchial catarrh is retained which forms the third period of pertussis in the aged. Complications are rare, and are almost always due to the irritation of the larynx and the strain of coughing. In rare cases brochopneumonia will occur during the disease, or may follow it. The treatment is as in infancy. A change of climate will sometimes hasten the cure. The medicinal measures usually given in infancy must be given in increased doses in senility, but vasoconstrictors must be avoided.

Mumps has been reported in the aged. The essential features of the disease are the same as those of earlier life but the orchitis which generally accompanies the disease in maturity does not appear in old age. The disease is usually mild, but a few cases have been reported in which death had occurred soon after cerebral symptoms, delirium and coma appeared. The treatment is purely symptomatic unless suppuration occurs when a free incision is indicated. The inunction with a 5 per cent. solution of oleate of mercury may shorten the inflammation.

Malaria is infrequent in the aged, either as a primary attack, or as a recrudescence of an earlier one. The disease differs

slightly in its symptoms from that of earlier life. The temperature is rarely above 103°, and where the first attack occurs late in life, there is no apparent enlargement of the spleen. Other symptoms, such as headache, malaise, aches in the bones, joints and back, thirst, anorexia, etc., may be aggravated. *Remittent fever* and *pernicious malaria* are extremely rare and the latter is almost always rapidly fatal. The ordinary intermittent fever is readily recognized by the regularity of its appearance in the tertian or quotidian form. In all cases Osler's dictum, "an intermittent fever that resists quinine is not malaria," holds good.

Chronic malaria or malarial cachexia follows repeated attacks of one of the acute forms and is usually fatal in the aged. The course and treatment of malaria is the same as in younger individuals. In the severe forms of remittent and pernicious fever quinine alone is of little service, except to reduce the temperature and this is rarely high in the aged. The quinine should be given in these cases combined with gr. 1/40 of arsenic three times daily or with methylene blue in 2-grain doses, while other symptoms should be treated symptomatically.

Yellow fever appears infrequently and when it does occur, it presents the same symptoms and takes the same course as in younger individuals. It is probable that the comparative rarity of yellow fever and primary attacks of malaria in the aged is due to the character of the senile skin which makes it less attractive to the pathogenic mosquito than the skin of younger individuals. The temperature in yellow fever is rarely high, and vomiting may be absent during the entire disease, while the icterus is not as pronounced as in younger individuals, probably due to the darker and more weather-beaten skin. Other symptoms such as headache, pain in the bones, joints, back and epigastrium, occur as in maturity. The stage of initial fever may be prolonged, the remission short and the reaction protracted. Most deaths occur during the stage of reaction and are due to profound impairment of the heart, kidneys, or liver. Some deaths are due to pulmonary edema following hypostatic congestion, or to general exhaustion. The diagnosis is readily made by the distinguishing features pointed out by Guitéras, the facies, early albuminuria and a slowing pulse, with a constant or rising temperature. There is a high hemoglobin percentage, and the blood count

shows an increased number of red cells and diminished leucocytosis.

The treatment of yellow fever is symptomatic and hygienic. Quinine has apparently no other effect than to reduce the temperature, and for this purpose it is the most reliable antipyretic we have. The bowels should be thoroughly cleared, using, preferably, castor oil; calomel should not be used. As intestinal antiseptics, we can employ salol and the sulphocarbolates. Nothing will stop the black vomit when it occurs, but the tendency to vomit can be diminished by small doses of cocaine. Should a reaction set in, the bile salts can be given to replace the diminished bile, hot fomentations over the kidneys and saline diuretics largely diluted should be employed in anuria, and heart tonics where the heart becomes weak, as it usually does in yellow fever. Spartein is the proper drug when the heart becomes weak and slow. Concentrated and predigested foods should be given throughout the disease and during convalescence.

Dysentery does not differ in its essential features from the dysentery of earlier life. Both the bacillary and the amebic forms occur in the aged, the two forms presenting the same symptoms. The bacillary form generally begins with chills and a slight elevation of temperature, after which the intestinal symptoms appear. These are frequent, small, painful dejections, abdominal cramps, tenesmus and straining and the passage of mucus and blood. There is rapid prostration and emaciation, great thirst and often rapid exhaustion. The disease is more fatal in the aged than in younger individuals and, unless controlled, a fatal issue may be reached in a few days. Since the introduction of the antidysenteric serum the death rate has been greatly lowered. The amebic form is usually slower in its onset than the other, it is more protracted and unless death results from toxemia, or from perforation of the bowel, it usually passes into the chronic form of dysentery. In this form there are remissions and exacerbations, the remissions being usually marked by alternating constipation and diarrhea. During an exacerbation there is a return of the usual symptoms of acute dysentery. During the remissions there may be a lienteric diarrhea, or there may be semisolid or fluid stools usually containing mucus, but rarely blood, while tenesmus

and straining may be absent. In this form of dysentery there is progressive emaciation, the individual becomes weaker and dies from exhaustion, the fatal issue being sometimes reached in a few weeks; more often, however, it does not ensue until several months or a year or more have elapsed. Complete recovery is extremely rare in advanced life. Numerous complications arise in the course of acute and chronic dysentery. These are due in part to the loss of blood and water, in part to the toxemia and septic infection and in part to the local destruction of tissue. The gangrenous process may extend through the wall of the bowel and cause perforation and speedy death. Hepatic abscess frequently follows amebic dysentery and abscess and gangrene in other tissues are occasionally observed. Septic inflammation may occur in any tissue and other bacterial diseases are sometimes associated with it. For the bacillary dysentery we have an antidysenteric serum which is curative in most cases but it is useless in the amebic form. In addition to the serum, other therapeutic measures are employed to produce a cessation of the discharges, relieve the distressing symptoms, and maintain the strength of the patient. The first indication is to clear the bowel with castor oil. After this has been accomplished the Cautani's enteroclysis solution should be employed as described under cholera. For the relief of the diarrhea we can use tannalbin, the sulphate or arsenite of copper, zinc sulphate, nitrate of silver, or any of the metallic astringents. Ipecac is probably the oldest and best drug for the control of the diarrhea. The dose is 10 to 15 grains every hour for four hours, given preferably in milk. As this may have an emetic effect, the patient must remain strictly in the recumbent position and 10 minims of tincture of opium should be given before starting the ipecac treatment. If there is pus in the discharges, salol or the sulphocarbolates should be given with the astringent. Belladonna given in a suppository will generally relieve the tenesmus and morphine may be used to relieve pain and insomnia. Magnesium sulphate often relieves the distressing symptoms. The various complications require appropriate medication. The dietary treatment is important, as improper food will aggravate the disease. The food should be liquid and concentrated, corresponding with the character of the stools. If the stools are

watery the food should be liquid. With semisolid stools give mushy food, and no solid food till stools are natural.

Plague does not occur in the United States except in isolated cases of immigrants coming from plague infested countries. In countries where it does prevail it is seldom found among the aged, and when they are attacked, it appears in a milder form than it does in maturity. According to Ortner the death rate is not higher, indeed it may be lower than in earlier life. This would tend to confirm the view that the aged organism is more resistant to infection than the younger.

Cholera when epidemic, attacks the aged as rapidly as younger individuals and is much more fatal, death occurring in almost every case, even when the symptoms are mild. The disease does not differ in its essential features from the disease in maturity. The period of incubation may be prolonged, but the onset and course of the disease are as in younger individuals. The discharges and vomiting soon induce profound prostration and collapse. In some cases collapse sets in before the choleric rice-water diarrhea has appeared; in most cases collapse occurs during the algid stage. Few aged patients survive this stage and most of those that do, succumb to a succeeding typhoid condition.

There is no specific treatment for cholera and all our efforts must be directed to counteract threatening symptoms. The vomiting may sometimes be checked by cocaine in 1/8- to 1/4-grain doses, chloroform in 3-minim doses or morphine in 1/8-grain doses, hypodermically. The usual treatment for the diarrhea is first a large dose of castor oil followed two hours later by 10 minims of tincture of opium. Cautani's enteroclysis solution for irrigation of the bowels should be used three or four times a day. This solution consists of tannic acid 2 1/2 drams, tincture of opium 30 minims, mucilage of acacia 3 ounces, to 4 pints of water. It is injected slowly at a temperature of 105°. For the muscle cramps, menthol and chloroform liniment, combined with massage, may give temporary relief. The usual heart tonics, camphor, caffeine, cactin, etc., must be used from the onset, reserving strychnine for the inevitable emergency during the algid stage or earlier. Dermoclysis and intravenous injections of normal saline solution are used when collapse sets in. Salol and the sulphocarbolates should be used as intestinal

disinfectants from the onset of the disease. Concentrated and predigested foods are necessary.

Cholerine, a mild cholera with slight muscle cramps, little or no vomiting and colored diarrheal discharges, is treated like the grave form.

Variola in the aged rarely follows the classical course. The prodromal period is more severe and prolonged. The cerebral symptoms simulate early meningitis, and may proceed to delirium and coma. There is always a weak, rapid pulse and often shallow rapid respirations. The initial symptoms are slight chills, rapid rise in temperature, prostration and pain in the lumbar region. The initial eruption is usually absent and the true eruption of variola appears as rose-colored macules, few in number and more scattered than in earlier life. Few of these macules proceed beyond the papular stage and fewer still become fully developed variola pustules. The disease is protracted in the aged, and in some cases vesicles do not appear until the tenth day and are converted into pustules three or four days later. The further progress of the disease is as in younger individuals, the whole course, however, being slower.

The disease is extremely fatal in the aged, the confluent and the hemorrhagic forms being invariably so. Many cases die during the period of invasion, while others succumb as soon as the stage of suppuration is reached. The complications include meningitis, pneumonia or bronchopneumonia, pleurisy, gangrene, bedsores, various forms of mucous inflammation, pulmonary edema, etc. The prodromal stage and the initial stage of variola, before the appearance of the eruption, is like the prodromal and initial stages of other acute infectious diseases and it is often impossible to differentiate between them. Some presumptive diagnostic points have been given, but until the appearance of the macules—which generally first show upon the forehead and wrists—a positive diagnosis is impossible. After their appearance there ought to be no further doubt, as the only disease giving a similar history of invasion and a similar rash being measles. This is readily differentiated by the milder symptoms, catarrhal and conjunctival inflammation, the more profuse rash and absence of lumbar pain, etc. In variola there is a sudden drop in the temperature as soon as the erup-

tion appears, while in all other exanthemata the appearance of the eruption is marked by a slight rise in temperature.

Treatment of smallpox is symptomatic and hygienic. There is no specific treatment and all that can be done is to treat symptoms, minimize the causes of complications and maintain the strength of the individual. The distressing or dangerous symptoms are the fever, cerebral symptoms (headache, delirium, coma), circulatory disturbance, pain, pruritus and exhaustion.

The fever can sometimes be controlled by antipyretics, especially acetphenetidin and others of the coal-tar products, but these are cardiac depressants and must therefore be combined with ammonia carbonate. The usual combination with caffein is irrational, as the caffein is a slow-acting heart stimulant, while the depressants act rapidly. Quinine is a safe antipyretic in all infectious diseases, but it is slow in action. The delirium should be controlled by the use of bromides and, if these fail, codein or morphine must be used. If the heart becomes weak, we must use cardiac stimulants, preferably caffein or camphor, leaving the more powerful stimulants, like strychnine, ether, strophanthus, etc., for emergencies. Nothing will take the place of cocaine as a local application for the intolerable itching that sometimes accompanies the eruption. For exhaustion we should proceed as in typhoid fever allowing, however, greater leeway in the selection of food. The hygienic and prophylactic measures are as in typhoid fever.

Varioloid occurs during an epidemic in persons who have been vaccinated. It is virtually a mild variola and as such can attack the aged as well as younger persons. The course of the disease is mild, the surface lesions rarely proceeding to the vesicular stage. The cerebral symptoms and exhaustion are, nevertheless, severe in the aged and may cause death. The treatment is similar to that of the graver disease.

Typhoid fever is infrequent in the aged. When it does occur its early manifestations resemble the early symptoms of pneumonia and it is often impossible to differentiate them without the Widal reaction or blood test. It generally begins after a prolonged prodromal malaise with slight chills, irregular fever and rapid prostration. The classical symptoms and course are rarely found in the senile cases. Instead of the

typical temperature curve there is usually an irregular temperature, sometimes remittent, sometimes intermittent, seldom going above 103°, more often remaining in the neighborhood of 102°; Instead of the usual progressive rise in temperature and increasing severity of symptoms for a week, followed by a week of maximum intensity of symptoms with a steady decline during the next week, the disease in the old runs an irregular protracted course lasting four or five weeks. The period of maximum intensity is about the end of the second week. The pulse shows some peculiarities, being often dicrotic, and, while in younger cases the rate is comparatively low, even when the fever is high, in senile cases it is generally high and may reach 120 or more with a temperature of 102°, and slight causes, such as a change of position, will cause a rapid rise of 20 or 30 beats per minute which drops again a minute or two later. The eruption is usually lighter in color, smaller, and scattered, and may escape detection altogether. Instead of appearing in successive crops the first crop appears at the end of the first week and may disappear in a few days or may persist throughout the disease. The abdominal symptoms frequently differ markedly from the classical symptoms as they appear in younger individuals. The spleen shows no enlargement. There is a progressively increasing tympanites which may set in during the first few days of the disease and which is more pronounced than in maturity; constipation is the rule, and the typical "peasoup" diarrhea is infrequent. There is some pain in the ileocecal region and occasionally in other localities about the abdomen. There are usually sordes about the teeth and tongue and the latter is dry, brown, often cracked. In severe cases there is extreme mental and physical depression, the patient is semicomatose or there is a low muttering delirium, the respiration is shallow, heart weak and rapid, and the whole appearance that of the prostration preceding death. In some cases there is a tremor of the hands, more often there is an unconscious picking at the bed-clothes, and twitching of the tendons, or else there is a complete relaxation of the limbs as in motor paralysis. The gravity of the symptoms increases until the end of the second week. Most senile typhoid fever cases succumb at this time, the prostration leading to collapse and death. In the cases that survive this period, there is a gradual

improvement, first seen in the cerebral symptoms. There is also a clearing of the tongue, a conscious effort to swallow, and the patient begins to sleep naturally. Later appetite returns and with it increasing strength. Constipation alternates with diarrhea, the meteorism disappears, the spots fade, and the patient is free from pain. Convalescence is slow and in most cases the complications that have occurred during the progress of the disease retard complete recovery for weeks or months after the disappearance of the disease. The most frequent complications are pulmonary edema following hypostatic congestion, bedsores, intestinal hemorrhage, cardiac exhaustion, bronchitis, pneumonia and perforation. Pulmonary edema and perforation are rapidly fatal. Intestinal hemorrhage is almost always fatal, as the aged individual cannot stand the loss of blood, and dies of exhaustion. Cardiac exhaustion can sometimes be overcome by the use of rapidly acting cardiac stimulants. A complicating pneumonia is generally fatal, either through the toxemia itself, or through pulmonary edema. The bronchitis that occurs in typhoid fever is usually purulent, which may lead to pulmonary abscess or gangrene or to a capillary bronchitis. There is occasionally an acute nephritis, and various ulcerative, hemorrhagic and degenerative conditions may result from the typhoid infection.

The diagnosis of typhoid fever in the aged is sometimes difficult unless the bacilli are found, or the Widal test is made. The clinical manifestations are often misleading, owing to the irregular course of the disease; lower temperature, light eruption, absence of splenic enlargement and frequent absence of the "peasoup" diarrhea. Mononuclear leucopenia exists in typhoid fever unless complicated by a disease in which leucocytosis occurs. Other diseases in which a leucopenia occurs are readily differentiated from typoid fever or do not occur in the aged. These diseases are measles, German measles, smallpox, mumps, malaria, tuberculosis, influenza, leukemia and pseudoleukemia. The leucocyte count is of importance, as it enables one to distinguish an early stage of typhoid fever from sepsis, pneumonia, appendicitis and meningitis.

The early clinical manifestations of typhoid may simulate pneumonia, sepsis or meningitis. The prostration seen in typhoid may be seen in pneumonia or any other acute

grave disease. In the serous inflammations—peritonitis and pleurisy—the mind is clear, in meningitis there is intense headache, photophobia, tinnitus, rapid prostration, and the mind is dull, unless there is delirium, when it is active. There is besides generally a history pointing to cerebral disease, while abdominal symptoms that are always found in typhoid fever are absent. The onset of pneumonia resembles the onset of typhoid fever. In the absence of a Widal test the early diagnosis based upon symptoms and physical signs must be determined by the presence or absence of the symptoms and signs of pneumonia. The cerebral symptoms are more pronounced in typhoid, and cough if not present at the onset of the disease does not appear during the first week, while in pneumonia it appears within the first forty-eight hours. After the second day the physical signs in most cases of pneumonia are sufficiently pronounced to determine the diagnosis. Other diseases occurring in the aged which begin with prostration, chills, fever and a profound malaise are sepsis, influenza and tuberculosis. The invasion of miliary tuberculosis is slow and never as severe as typhoid. The early bronchial symptoms of influenza do not appear in typhoid until the end of the first week; the tongue is red and moist, in influenza the skin is reddened, there is often a herpetic eruption and generally a copious purulent or mucopurulent expectoration. The early differential diagnosis between sepsis and typhoid in the aged is often difficult and even if the eruption appears it is not always a certain pathognomonic sign as roseate macules are occasionally observed in sepsis and the beginning of typhus. The diagnosis of sepsis is often based upon the presumptive signs of a pronounced initial chill, pains in the bones, herpes, hemorrhagic macules, rapid pulse and respiration, while rapid profound prostration with cerebral symptoms, roseate papules, and eventual intestinal hemorrhage point to typhoid. Any of these presumptive symptoms and signs may occur in either disease and we must often make a diagnosis by the prominence and number of symptoms found most frequently in either disease. In some cases a positive diagnosis cannot be made without examination of blood, urine and feces. Typhoid may also be mistaken for paratyphoid, typhus or epidemic cerebrospinal meningitis. The last two are very rare in the aged, appear only in an epi-

demic form, and proceed to a very grave condition within forty-eight hours. Cerebrospinal meningitis presents a pathognomonic pain along the spine with stiffness of the muscles of the neck. Typhus begins with a chill, there is high fever, rapid and profound prostration and the eruption is scattered over the trunk and not confined to the abdomen. The eruption consists of rose-colored macules with hemorrhagic centers. In paratyphoid, which is a rare disease, there is an initial chill, the cerebral symptoms are milder, while the intestinal symptoms are more pronounced. There is usually early vomiting and diarrhea, frequently herpetic eruptions, irregular temperature and the whole course of the disease is milder.

The prognosis of typhoid fever in the aged is always grave, even in cases where the symptoms are mild. The chief sources of danger are the prostration leading to collapse, pulmonary edema following hypostatic congestion, intestinal hemorrhage and perforation, and pulmonary and renal complications. Protracted cases produce general exhaustion and a relapse is almost always fatal. Many deaths are due to complications other than pulmonary and renal involvement.

Treatment.—There is no positive method of aborting, shortening or curing typhoid fever, the typhoid vaccines being still experimental, and therapeutic measures must be directed toward amelioration of symptoms and prevention of complications. The most important of the therapeutic measures in maturity is hydrotherapy applied in the form of cold baths given according to the Brand or Baruch method. In senile cases a cold bath is an extremely dangerous experiment. The shock may produce collapse, while if the temperature of the bath has been gradually lowered, there may be no reaction in spite of friction, hot water bottles, hot stimulating drinks, etc.

If the rectal temperature is 103° or above, cold sponging may be tried, but if the first application of cold water (never ice in the aged) produces a shock it must be discontinued or tepid water substituted. The sponging can be repeated every two or three hours, but the patient must be moved as little as possible, and abdominal manipulation must be avoided. The usual practice of beginning the treatment of typhoid fever by giving repeated small doses of calomel can serve no other useful purpose than to produce catharsis. It has no influence upon the disease.

If there has been constipation a single dose of castor oil to which 5 grains of salol, betanaphthol, or soda sulphocarbolate and 2 grains of the bile salts have been added, will act better than calomel. If there is diarrhea a powder containing dionin 1/6 grain, salol 5 grains and bismuth subnitrate 10 grains should be given, and repeated if necessary in three or four hours. Salol and the sulphocarbolates (Waugh Abbott formula) can be used as intestinal antiseptics throughout the disease. In hyperpyrexia quinine will give the most permanent results, but if quick action is necessary, as in delirium due to high temperature, we must fall back upon the coal-tar preparations, preferably acetphenetidin or antipyrin. A temperature of 104° or more in an aged patient generally points to a complicating infection. If the usual antipyretics do not reduce the temperature and there are pronounced cerebral symptoms, it may be necessary to resort to the cold bath notwithstanding the danger of shock and collapse. The bath should be followed by friction and hot water bottles to the feet. For insomnia, urethane, veronal, sulphonal or trional can be used, and opium only if the other remedies fail to produce sleep. The carbamate group is rather safer and more reliable than the methane group. It is of the utmost importance to maintain the strength of the patient. This is accomplished partly by appropriate diet and partly by drugs. The drugs to be used for this purpose are small doses of strychnine, caffeine and carbonate of ammonia. Digitalis is always dangerous and strophanthus should be used only if the heart becomes weak and rapid. In a weak and slow heart spartein should be used in 1/2-grain doses every three hours until there is response. Exhaustion of the heart is a constant danger and requires prompt treatment. When this sets in we must resort to hypodermic injections of strychnine, ether and camphor, and give internally, brandy and hot coffee. The head should be lowered and hot water bottles placed to the feet.

The position of the patient should be occasionally changed to prevent hypostatic congestion. It is impossible to guard against intestinal hemorrhage or perforation, which sometimes occur in spite of every precaution. Absolute rest, giving the patient an opiate if necessary, is the only safe measure that can be suggested. Subcutaneous injections of a 2 per cent.

solution of gelatin have been tried in hemorrhage and success is reported. Ergot and adrenalin are extremely dangerous in the aged on account of their vasoconstrictor effect upon the whole circulatory apparatus, nevertheless, if the bleeding continues, a hypodermic injection of 2 grains of ergotin and 1 grain of stypticin can be tried. High enemata of starch, gelatin and hemostatics have been suggested. When we remember that it is virtually impossible to get the clyster past the ileocecal valve and that the hemorrhage almost always comes from a typhoid ulcer in the ileum the uselessness of attempting local medication by way of the rectum must be apparent. A more rational treatment of intestinal hemorrhage would be by means of the metallic astringents, zinc or copper sulphate given by mouth, but while the hemorrhage may be controlled, the danger is from shock and exhaustion which may follow the loss of even a very slight quantity of blood. There is also a danger from the irritation of these salts upon the ulcers. There is no known method of combating perforation. Abdominal section has been recommended but no cure has been recorded. Other complications require the ordinary treatment for such. Bedsores can be avoided if the skin is kept dry and the pressure points are protected. The care of the typhoid fever patient is more important than drug treatment and the diet alone may change the entire aspect of the disease. In the selection of food, two factors must be considered, to maintain strength and to prevent irritating matter from reaching the intestinal lesions. The latter factor presents peculiar difficulties as it is hardly possible to arrange a dietary which will not contain refuse matter, or matter liable to undergo fermentation or decomposition in the bowel. The safest food is fresh milk, but the amount necessary to support the strength of an aged person, three to four pints daily, imposes excessive work upon the circulatory system. To avoid the excessive quantity of fluid, the condensed or evaporated milk should be used. If the milk diet becomes objectionable, its taste can be masked by the addition of salt, coffee or chocolate, or one of the prepared foods may be substituted temporarily or added. The foods containing a large percentage of alcohol should be avoided and likewise foods consisting principally of unconverted starch and those containing a large amount of lime. During the first

week, while assimilation is good, we can use concentrated foods, etc. After the first week, or if assimilation is poor and undigested food particles appear in the feces, the food should be partially converted or predigested. The present-day tendency is to permit greater latitude in the variety of foods, but if the previously recommended food substances can be taken, there will be less danger of intestinal irritation. There are numerous simple articles of food, such as thin barley gruel, albumin water, malt extract and gelatin, which are unobjectionable and which may be occasionally given, but they contain comparatively little nutritive value. When there is extreme exhaustion and distaste for food, only the most concentrated foods should be used. Coffee may be given throughout the disease. Solid food should not be permitted until at least ten days after the temperature has become normal. Other hygienic measures, as fresh air, sunshine, rest, quiet, the avoidance of motion except the occasional shifting from side to side to prevent hypostatic congestion, are self-evident. The patient should not be permitted to exert himself, to arise, move, turn, talk much, etc. A bed pan which will slide easily under the body, must be used.

The mouth, tongue, teeth and lips should be regularly cleansed with an alkaline antiseptic solution, preferably one containing formaldehyde, or one of a solution of peroxide of hydrogen or permanganate of potash. It is almost unnecessary to caution the attendants about the thorough disinfection of the discharges, fecal, urinary and salivary, the bed pan and cuspidor, the clothing and bedding and everything that had been used about the patient. The physician himself is frequently the carrier of the infection and this he can avoid only if he will change his clothes before seeing the next patient and disinfect the exposed ones. The recently introduced antityphoid serum is apparently a reliable prophylactic.

Paratyphoid fever presents apparently no marked differences between that of maturity and that of senility. The few cases reported give symptoms resembling a mild typhoid and in most cases bacteriological or Widal reaction tests are required to determine the diagnosis. The disease is treated as is typhoid fever.

Typhus fever is relatively more frequent and more fatal in

the aged than in maturity. It is, however, rarely met with in the United States and then almost exclusively in recent immigrants coming from countries in which it is endemic. In the aged the period of incubation is prolonged, the disease develops slowly and the characteristic eruption may not appear until the tenth day or later. There is, however, the sudden onset with a pronounced chill followed by a fever which may reach 104° on the third or fourth day. The temperature is irregular, remittent, or continuously high. The pulse is rapid and weak. Cerebral disturbances occur early and delirium, with subsultus tendinum and coma, may appear during the first week. The initial eruption of red macules soon shows a dark hemorrhagic center. In some cases the eruption begins with petechiæ, in others the macules almost immediately become hemorrhagic. These are rapidly fatal cases. There is extreme prostration from the onset of the disease and the functional activity of all the organs is impaired. Sordes and other typhoid symptoms appear earlier and are graver than in typhoid fever and the same complications that may occur in typhoid may also occur in typhus. Hypostatic congestion is one of the most frequent ones and is fatal. The early diagnosis of typhus fever is difficult, as typhoid fever, cerebrospinal meningitis, relapsing fever, smallpox, measles, sepsis and pneumonia may all begin with a chill, high fever and prostration, following a period of malaise. Relapsing fever can be excluded from consideration as it does not occur in the United States and has a specific spirocheta in the blood which is recognizable from the onset of the disease. Measles presents neither the distinct chill, high fever nor prostration of typhus. The pain and contractions of the spinal muscles occurring in cerebrospinal meningitis are absent in typhus. In variola the eruption begins as discrete papules on the forehead and wrists and there is a fall in the temperature and clearing of the mind as soon as they appear. Before the appearance of the eruption there is no diagnostic sign by which the two can be differentiated although the prostration is not so severe in smallpox nor is the mind dulled. In hemorrhagic variola the initial symptoms may be fully as severe as in typhus, but the eruption is vesicular and there is also an eruption upon the fauces. In pneumonia the cough and physical signs may be detected on the second day. The pres-

ence of the pneumococci will establish the diagnosis. Typhoid fever may give the same severe initial symptoms as typhus, or the latter disease may give mild initial symptoms. In either case no definite diagnosis can be made until the appearance of the rash, which in typhus may be seen on the fourth or fifth day, though in the aged it appears later, and the bacteriological finding of the typhoid bacillus. In sepsis an early diagnosis can usually be made by the presence of the pyogenic germs. In most contagious diseases the existence of an epidemic simplifies the diagnosis.

Treatment is purely symptomatic and demands, primarily, the support of the patient's strength. There being no specific intestinal lesions as in typhoid fever, greater latitude is permitted in the selection of food, which should be as concentrated as possible, and any indication of intestinal disorder, as evidenced by sour or foul-smelling stools, or the presence of particles of undigested food, should be met by a thorough catharsis, followed by an intestinal antiseptic, and a milk or predigested food diet. The medicinal treatment is the same as in typhoid fever.

Influenza occurs rather frequently, the advantage of greater resistance being more than counterbalanced by the presence of chronic bronchitis, the impaired mucous membrane forming a fertile field for the propagation of the pathogenic bacilli. The disease in the aged is usually of a toxemic respiratory type, rarely the nervous form, still more rarely the gastrointestinal type. There are rarely typical cases of any of these forms of the disease, most cases presenting symptoms of all types, the toxemic and respiratory symptoms predominating. There are no marked differences in the symptoms between maturity and senility. The temperature is usually low, rarely over 102°, frequently it ranges between normal and 100°. Owing to the atrophy of the nasal mucous membrane the rhinitis may be absent but conjunctivitis may be marked. The disease begins with the usual mild chills followed by elevation of temperature, headache, pains in the extremities and back. A pharyngitis is noted, followed by a laryngitis, the catarrhal inflammation proceeding downward into the trachea and bronchi. Facial herpes occurs frequently and the face is usually flushed, sometimes in patches. The disease itself is not grave, but the frequency of pulmonary complications, especially lobular pneu-

monia and pleurisy, makes it one of the more serious diseases of old age. Owing to the mildness of the initial symptoms of the bronchopneumonia, that complication is frequently overlooked until near the fatal end. (See Senile Pneumonia.) The earliest symptom of bronchopneumonia is usually an irregular rise and fall of the temperature, but this may not be noticed unless the temperature is taken every two hours after the first rise is noted. Circulatory disturbances, evidenced by weak cardiac impulse, weak pulse, arrhythmia, dyspnea and cyanosis, occur frequently, especially during the period of convalescence. Cerebral and nervous complications are infrequent with the exception of trigeminal neuralgia. Some cases of lobular pneumonia appear without the initial coryza, and laryngitis, and the diagnosis can be made only by finding the influenza bacillus, and cases have been reported giving cerebral symptoms alone but showing the bacilli in the cerebrospinal fluid.

A positive diagnosis can be made only when the pathogenic germs are found. In the absence of these findings, the disease may be mistaken for a simple cold, although in the latter attack the acute invasion, prostration, neuralgia and herpes are absent or mild. The coryza, and early bronchitis will distinguish it from meningitis, tuberculosis and typhoid.

Ortner describes under the name *chronic influenza* a type which is protracted or recurrent and where the bacilli can be found for a long period in the sputum and nasal secretion. It occurs in aged emphysematous individuals, either following an acute attack, or coming on with mild symptoms of malaise and coryza. The symptoms may persist for months and the bacilli are found for a long time after all symptoms have disappeared.

The treatment of influenza is symptomatic and hygienic. French physicians use colloidal metals by inunction or by subcutaneous or intravenous injection as curative agents in infectious diseases generally, but this mode of treatment is still experimental. For the relief of symptoms the measures useful in earlier life can be employed, with due regard, however, for the degenerated state of the tissues. Antipyretics are rarely required. For the neuralgia, distressing cough without expectoration, or with scanty tenaceous mucus, for insomnia, dyspnea, etc., the same treatment is required as in simple neuralgia, bronchitis, cardiac asthma, etc.

ACUTE ENDOCARDITIS

Etiology.—Contrary to the generally accepted view that acute endocarditis is probably of bacterial origin in all cases, we find that but few cases occurring in old age follow a bacterial disease, while even in younger life endocarditis has been found in most cases of fatal chorea. We must either assume that such diseases of the fourth group as diabetes, gout, cancer and chronic nephritis during which valvular disease frequently develops, are bacterial, or else we must drop the assumption that acute endocarditis is always of bacterial origin.

Acute endocarditis is due to inflammation of the endocardium produced by some irritating constituent of the blood. This may be bacteria or bacterial or other toxins, or else the abnormal constituents found in the blood in diabetes, gout, nephritis, etc. The bacterial endocarditis is rare in the aged as bacterial diseases producing endocarditis are infrequent at that time of life. The simple non-bacterial form is rarely recognized, the assumption of its existence being usually based upon the production of a valvular lesion in the course of the causative disease.

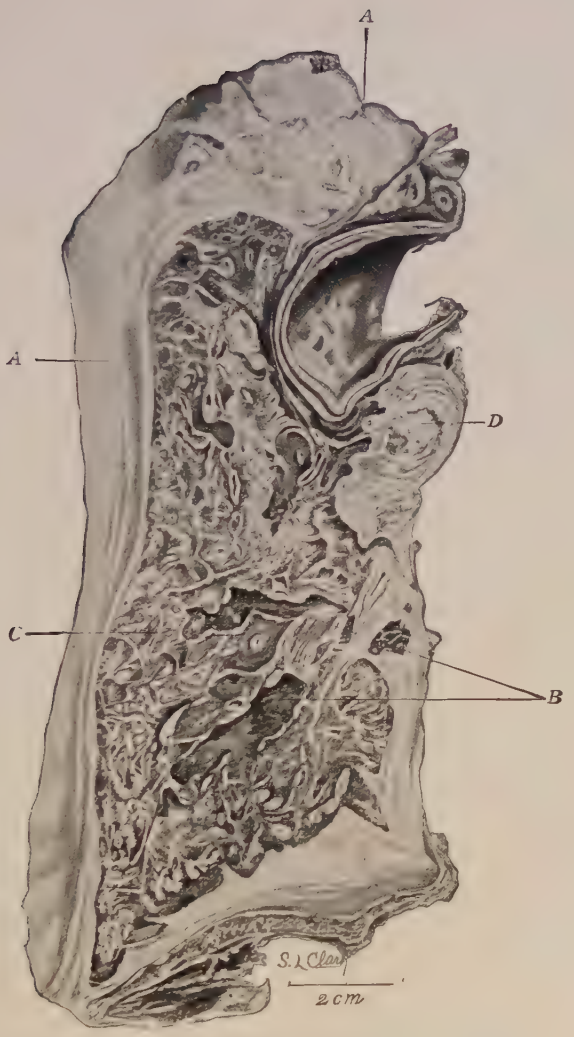
Pathology.—In the bacterial form there is a deposit of fibrin in which leucocytes and blood plates are imbedded upon the valves or less frequently upon the cordæ tendinæ or other parts upon which the blood impinges. This is followed by a proliferation of endothelium and subendothelial connective tissue into this deposit, the whole forming the so-called "vegetations," fringes or warty excrescences from 1/30 to 1/4 inch in thickness, continuous with the adjacent tissue. In rare cases the vegetation is attached to the base by a pedicle. These vegetations interfere with the free action of the valves, sometimes they contract, producing marked valvular deformity. During the continuation of the disease the vegetations are covered with a layer of fibrin which affords a lodgment for pyogenic bacteria and if an invasion of such bacteria takes place the disease becomes an ulcerative or malignant endocarditis. In this case the vegetations may soften, ulcerate and suppurate, necrosed tissue is thrown off and carried in the blood current as emboli, usually plugging some vessel in the brain, liver, spleen, kidneys or other organ. This latter con-

dition is, however, extremely rare in the aged, as the aged invariably succumb within a few days after the septic infection. Fragments from non-septic vegetations may be torn off and cause emboli and infarctions in distant parts.

In the non-bacterial endocarditis there is a thickening of the endothelium with deposit of fibrin, but no organization into vegetations. The action of the valves is, however, always interfered with. In most cases occurring in the aged there are at the same time senile changes in the endocardium (see Senile Endocarditis) and only the presence of the fibrin deposits distinguishes the acute inflammatory condition from the non-inflammatory senile degeneration.

Symptoms.—In the non-bacterial variety of acute endocarditis, before the valvular lesions give their distinctive symptoms, the only symptom that might attract attention is irregular heart action. Even this usually escapes notice until the valvular murmur appears. When the irregular heart action is so marked as to be noticeable it is usually attributed either to the causative disease or else to the senile changes in the heart. There is no pain or elevation of temperature connected with this variety of endocarditis.

In the bacterial variety the earliest symptom is a rise in temperature not due to any apparent change in the character of the causative disease. The rise may be two or three degrees. About the same time the heart action becomes accelerated and irregular, there is a feeling of discomfort about the heart and the circulation is disturbed. These symptoms, which are usually mild, are followed by symptoms of valvular incompetency, occasionally stenosis, the mitral valve being usually first involved. The later symptoms are those of the valvular lesion. In the malignant or ulcerative form of acute endocarditis there is a sudden rise in temperature of two or three or more degrees, marked mental and physical depression, sweating, irregular heart action rapidly followed by the valvular lesion and its symptoms. In the aged death usually occurs in a few days without the occurrence of embolism, the most frequent complication in younger individuals. There are several types of this variety of endocarditis, typhoid, septic, meningeal and a type in which the cardiac and circulatory disturbances are most prominent. The rare cases that occur in the aged present



Lung, Chronic Interstitial Pneumonia, Bronchiectasis, Hyaloserositis, and a Terminal Catarrhal Pneumonia Resulting from Concurrent Infection by the Tubercle Bacillus and Pneumococcus. (From Coplin's "Manual of Pathology.") *A, A.* Greatly thickened pleura. *B.* Dilated bronchi. *C.* One of many strata of fibrous tissue irregularly transversing the organ. *D.* Large caseous lymph-node near hilum of lung, and immediately adjacent to the aorta, a section of which is shown just above. The aorta is the seat of slight atheroma.

typhoid symptoms. These cases are invariably fatal. The non-malignant forms in the aged become chronic and follow the course of senile endocarditis.

Treatment.—No form of treatment has been of the slightest avail in the malignant form of the disease. For other forms rest is of the utmost importance. Cloths wrung out in cold water and placed over the heart will relieve temporarily the irregularity and the feeling of discomfort in the organ. As long as the underlying cause persists the irritation and inflammation will persist and continue to do irrepairable damage. The further treatment is such as has been indicated under senile endocarditis and the valvular disease that had been produced. Various drugs have been recommended, such as the iodides to promote absorption of the lymph, mercury in various forms to prevent the deposit of fibrin, alkalies to increase the alkalinity of the blood, aconite and veratrum to weaken the force of the heart, etc. They are rarely given in time to be effective.

INFECTIOUS PNEUMONIA

It has already been stated in the chapter on senile pneumonia that faulty nomenclature and other causes have produced confusion in the conception of the various pathological states included under the term "pneumonia." In this work all pulmonary inflammations are divided into two classes, infectious and non-infectious pneumonias. In the non-infectious class are placed inflammations due to irritation, extension of non-infectious inflammations, and secondary inflammations not due to pathogenic germs. In the infectious class are included those inflammations which are caused by pathogenic germs.

Etiology.—The most frequent cause of *infectious pneumonia* is activity of Frankel's pneumococcus. Other germs that are known to cause pulmonary inflammation are the Friedländer bacillus, streptococcus, staphylococcus, micrococcus catarrhalis, the diphtheria, influenza, typhoid and colon bacilli and the meningococcus. It is believed that other germs are also able to cause the disease, as it occurs occasionally in the course of measles, whooping cough, typhus, variola and other presum-

ably germ diseases. The senile organism is more resistant to germ activity than the organism in earlier life, but the resisting power may be lowered through general debility. In most cases a fertile field is prepared by a previous disease or by a momentary perversion, as, for example, when inhaling cold air, a temporary hyperemia is produced in order to raise the air to the body temperature, this hyperemic surface becoming a suitable field for germ development. Local traumas leave points of exposure for the entrance of the pathogenic germs, while the shock of traumas generally reduces the vitality of the body and lowers its resisting power to germ development. The irritation produced by the inhalation of dust or noxious vapors causes local hyperemia, or a catarrhal condition which favors the bacterial activity upon which the disease depends. Passive hyperemia, bronchitis, pneumokoniosis, and interstitial pneumonia, all supply suitable fields for the propagation of the germs. While the germs generally reach their field by inhalation they may reach their localization in the lungs through the lymph and blood channels.

A pure pneumococcus infection is rare in old age and therefore a classical acute lobar pneumonia is infrequent. It is almost always a mixed infection in which the pneumococci and streptococci predominate. In many cases no pneumococci are found, and in cases where pneumonia occurs as a complication of another infectious disease the germs of the primary disease predominate, or are alone. Most cases in the senile are secondary.

Pathology.—The classical stages of acute lobar pneumonia are rarely found in the aged. The disease may be localized or diffused. Upon section during consolidation, the surface appears dark red, smooth and moist, exuding a bloody serum. Gray hepatization is very rare, the patient seldom surviving the stage of red hepatization, and where recovery does take place, resolution begins during this stage. In diffused pneumonia, hyperemic areas are scattered throughout the lungs, frequently in the upper lobes or in the upper part of the lower lobes. The microscopic appearance depends mainly upon the type of the predominating germs. In cases where the pneumococci predominate, the alveoli are filled with a fibrinous exudate. The influenza bacilli cause greater destruction of the epithelial cells, and their debris may completely fill the alveoli. If pyogenic

germs are present, pus cells will be found. In most senile cases the alveoli contain principally serum and broken-down epithelial cells, some fibrin, red blood cells, leucocytes and occasionally pus cells. The capillaries are swollen and engorged. Incidental pulmonary lesions such as caseous degeneration due to tuberculosis, abscess and gangrene due to pyogenic bacteria, interstitial edema, etc., are found occasionally.

Symptoms.—The usual sudden onset of *acute lobar pneumonia*, with a chill followed by high fever, is infrequent in the aged. When this does occur there is almost always a profound pneumococcic infection, with rapid prostration, and a fever ranging from 102° to 104°. Within a day or two there is a distressing cough, and some time later blood-streaked expectoration appears. The sputum is scanty and tenaceous; in some cases, however, it is entirely absent. Many senile patients will swallow the expectoration unless watched. Pain is usually slight. In this form of infectious pneumonia the disease is grave from the onset, and prostration is rapid, the breathing is shallow and rapid, but not panting as in younger persons; dyspnea is not marked, but cyanosis sets in early and cerebral symptoms are pronounced. The heart at the onset acts with increased force and rapidity, but it soon becomes weak and grows weaker as the prostration increases. Delirium sets in early. There is usually constipation, the urine is diminished and albuminous and may contain casts. The amount of urea is diminished. In acute lobar pneumonia in the aged, death may occur during the first or second day, and is rarely delayed beyond the eighth day. If improvement does take place the fever gradually subsides, the expectoration becomes more profuse and the mind becomes brighter. The face, which has usually a dark flush over the malars, resumes its normal appearance. Recovery is by lysis. A sudden drop in temperature occurs before dissolution, while a copious watery expectoration, with increasing dyspnea, indicates pulmonary edema.

In most cases of infectious pneumonia in the aged the disease develops rapidly but not suddenly. Where it occurs secondarily to another disease, the symptoms of the primary disease are aggravated, there is besides a rapid rise in temperature, dyspnea, and rapid shallow breathing, followed by cough and expectoration, which after a day or two becomes blood-streaked

and may be purulent. In some cases the system had been so weakened that the patient is unable to cough while the mind may be too impaired to realize the import of the local irritation produced by the secretion in the vesicles and tubes. In such cases cough and expectoration will be absent, but the increasing amount of secretion will lessen the aerating surface, the dyspnea will increase, cyanosis sets in and the respiration becomes more rapid. In every case of pneumonia, whether infectious or non-infectious, primary or secondary, localized or diffused, there is rapid prostration, loss of appetite and mental depression. There is generally some pain when coughing or when taking a deep inspiration. If the disease is localized, the pain is located in the affected part, but where there are scattered areas of consolidation, the pain on inspiration is most severe where the affected part of the lung in expanding rubs against the pleura,—while the pain on coughing is most severe in those areas in which the consolidation has been most complete, *i.e.*, in the parts first involved. There may also be pleuritic pain on coughing. Pain is not felt when cerebral symptoms appear. A pneumonia following a hemorrhagic infarct of the lung presents a blood-stained watery expectoration, but this condition is seldom found in the aged. The physical signs depend upon the type. There is dulness upon percussion over the affected site, but this dulness may be completely masked by an area of emphysema over the consolidation. In the localized type, dulness at the base points to a hypostatic pneumonia which may be infectious or non-infectious. The usual sites of a localized pneumonia in the aged are the apex or base of the upper lobe, the apex of the lower lobe or the side of the upper or middle lobe. The dulness is then found either in the supraclavicular space, or in the interscapular space, or below the axilla. In the diffuse type, small areas of dulness may be found over all parts of the lung. An important sign, both of the disease and its locality, is that of crepitation, heard at the end of inspiration and at the beginning of expiration. Owing to the senile anatomical changes in the chest wall, we do not find any difference in expansion and owing to the frequent presence of an old bronchitis râles may be heard in all parts of the chest.

When resolution sets in the symptoms abate, but in the

diffuse type new foci of infection may occur, while old areas of consolidation clear up, and thus we may get an irregular temperature with remissions and intermissions; periods of improvement with relapses, and with changing areas of infection. The pneumonia may be then prolonged for several weeks, the system becoming constantly weaker and the patient finally succumbing from general exhaustion, cardiac exhaustion or pulmonary edema. During the initial fever the heart action is more powerful than normal, but it rapidly weakens, partly as a reaction from the excessive activity, partly through impaired pulmonary circulation (the right heart being weakened and dilated), and partly through the toxemia which alters the circulating medium and interferes with the vasomotor regulation. These circulatory changes cause passive congestion of the liver, spleen and kidneys, with consequent impairment of their functions. The urine is diminished in quantity, the amount of urea and chlorides is lessened, while albumin, casts, an excess of uric acid and red and white blood cells are usually present. When resolution sets in the urine becomes normal unless a nephritis had developed. The most prominent and most serious indication of hepatic disturbance is icterus.

The blood in infections pneumonia shows a leucocytosis, with increase in fibrin. When resolution sets in a proteolytic ferment appears in the blood. Nervous and cerebral symptoms usually appear early such as headaches, insomnia, later delirium and finally coma, diminished reflexes, neuritis, etc.

Pneumonia is the most fatal disease of the aged. This is partly due to the fact that the onset is usually so mild that the disease is far advanced before it is recognized. It is only in the forms that are rare in old age, such as acute lobar pneumonia and general pneumococcic infection that pronounced symptoms appear from the onset of the disease, and these forms are usually virulent and fatal. It is the most frequent of all complications and the hypostatic form is a constant menace in every case in which a patient is confined to his bed. In many cases the physical signs can be found before there are any suggestive symptoms and it is therefore necessary frequently to percuss the back of the chest in order to determine any foci of dulness present. In some cases, where the only suggestive symptom of pneumonia is rapid prostration, we can get a history of exposure

which may show the inhalation of cold or rarified air or of noxious vapors; or the patient may remember that a bit of food went "in the wrong way," thereby producing a deglutition pneumonia. More often there is a history of another disease or traumatism and the examination of the chest will explain why the symptoms suddenly became aggravated. Cases may recover when seen early, but where pneumococci predominate, or if the disease is secondary to typhoid fever, death is almost inevitable. While the prognosis is unfavorable in every case of pneumonia, whether infectious or non-infectious, there are cases offering a fair chance for recovery. These are the inhalation pneumonias and those pneumonias that are due to other germs than the pneumococci. Rapid prostration diminishes the chances for recovery. Purulent or mucopurulent expectoration indicates the presence of pyogenic germs which may cause abscess or gangrene of the lung. The only diseases that may be mistaken for pneumonia are pleurisy, bronchitis, tuberculosis and typhoid fever. In early pleurisy there is absence of percussion dulness, and of rapid shallow respiration; there are friction sounds, but no râles. In pleurisy with effusion, the change in the level of percussion dulness upon change in position will distinguish it from the graver disease. In bronchitis there are no areas of dulness, and there is generally a history of previous cough and expectoration, the expectoration being thinner and more profuse than that of pneumonia. There is no prostration or pain. Tuberculosis is slow in its progress and the sputum contains the pathogenic bacilli. If there is any doubt about the differential diagnosis, it can be cleared up by the microscope and by the tuberculin test. The differentiation between typhoid fever and pneumonia depends upon the bacteriological examination of the sputum, blood and feces, and upon the Widal test.

Treatment.—The treatment of infectious pneumonia follows, in the main, the lines laid down for the treatment of senile pneumonia. In addition to these measures, which are intended to relieve symptoms and prevent the most frequent causes of death, serum therapy should be employed. Infectious pneumonia in the aged is almost always a mixed infection and the antipneumococcic serum has little effect upon it. Serum therapy is worse than useless if it is not based upon a bacterio-

logical examination. In all cases in which the symptoms may be ascribed to two or more forms of bacteria it is necessary to make such examinations before using any serum, vaccine, bacterin or any other bacterial product. There are various combinations of sera and vaccines on the market and the selection of the appropriate combination depends upon the bacteriological examination of the expectoration, or of the blood. Wherever possible, an autogenous vaccine should be used in preference to stock vaccines.

Some incidental symptoms may occur in infectious pneumonia that do not occur in the non-infectious senile form. For the fever we can use quinine or tepid sponging, never the coal-tar products or cold baths. Creosote is of service if the cough is distressing. The pernicious practice of giving narcotics is probably responsible for many deaths in pneumonia, by further weakening the respiratory centers, and by allaying the irritation caused by the mucus, which is so necessary to arouse the reflex action of coughing for the removal of the secretion. If the expectoration is thin, yet the patient cannot bring it up, senega should be given, but if there is a scanty tenaceous expectoration, muriate of ammonia should be employed.

The hygienic measures are the same as those of senile pneumonia.

TUBERCULOSIS

Tuberculosis in the form of fibroid phthisis occurs more frequently in old age than in earlier life. The disease is rarely a primary infection in the aged, being in almost every case a recrudescence of a disease that had been apparently arrested years before. It is possible that in some cases the infection has occurred at a time when the body was able to resist its pernicious activity and that in the course of years the resistance was lowered and the latent bacilli became active, or that late in life the virulence of the germs had increased. The acute or general tuberculosis is rare in advanced life and, while local infection may occur almost anywhere just as in younger individuals, the usual location is in the lungs where it produces a fibroid degeneration.

Fibroid Phthisis

This is the usual form in which tuberculosis appears in the aged. The disease is slowly progressive and may exist for years before the symptoms are sufficiently severe to cause the patient to seek medical aid, or before a particle of blood-streaked sputum attracts his attention. The typical symptoms, as they appear in pulmonary tuberculosis in early life, do not occur in the aged. The temperature is normal. There are no night sweats, no rapid emaciation, rarely secondary tuberculous diseases. There is, however, dyspnea, cardiac palpitation or arrhythmia, and the face becomes dusky or cyanotic; but these symptoms are usually attributed to the heart and impaired circulation. There is usually a cough, and the expectoration may be profuse or scanty. In some cases there is little cough or expectoration, the slight coughing being due to the irritation produced by a secretion of mucus of atrophic bronchitis. The expectoration is occasionally blood-stained, especially after severe attacks of coughing. Pulmonary hemorrhage, in which a quantity of blood is lost, may be due to erosion of a blood-vessel, to excessive or sudden strain or to a sudden rise in blood pressure, and cases have been reported in which there was a rupture of a varicose vein in the trachea and rupture of a vessel in a bronchus while straining at stool. A profuse hemorrhage is fatal.

Late symptoms are cachexia and emaciation, persistent dyspnea, cough and expectoration, the latter purulent or mucopurulent and occasionally blood-streaked and a sinking of the chest walls over the diseased part of the lungs. The supraclavicular and infraclavicular spaces are deeply depressed, most markedly so on the affected side. While the respiratory movements in old age produce a rise and fall of the rigid chest walls instead of an expansive movement, there may be noticed during inspiration a retraction of the intercostal spaces on the affected side and a bulging on the unaffected side. If both sides are affected the intercostal spaces of both will be retracted. Percussion gives dulness over the affected area, the percussion sound during deep inspiration being shorter, and almost flat. There are, however, many sources of error in percussing a senile chest with fibrous phthisis. Pneumokoniosis, senile emphysema, the atrophied lung, pleuritic adhesions, areas of hyperemia, and old cavities, all tend to modify the percussion note.

The auscultatory signs are often difficult to interpret as there may be all varieties of râles and breathing, friction sounds and murmurs. Expiration is usually prolonged and a tuberculous click can often be heard over a cavity. It resembles a single coarse bubbling râle but is louder and deeper and is often felt by the patient. It is pathognomonic of a cavity. The character of the râles depends upon the character of the bronchial catarrh, this condition being almost always present, but other pathological râle-producing conditions may also exist, which give no other symptom than these râles. Radiography gives valuable information, light areas indicating cavities, dark areas indicating consolidation, induration or growths. In all cases the finding of the bacilli in the sputum establishes the diagnosis, although they may be absent at one examination and present at the next. The various tuberculin tests can be used, but a positive reaction may be due to a former cured disease. There are frequent complications arising from extension of the tubercular process. Hoarseness and dysphagia are generally due to laryngeal tuberculosis, pain may be due to irritation of the pleura, to pressure upon a nerve or to a neuritis. There is occasionally a tubercular pericarditis, rarely with effusion, and tubercular ulcers of the intestines may occur especially in those who swallow the sputum instead of expectorating it. Liver and spleen are occasionally enlarged when the circulation is impaired and passive congestions result in the viscera. The presence of the bacilli is the only distinctive feature by which this disease can be distinguished from fibrous pneumonia.

In the treatment of fibroid phthisis in the aged the same general hygienic measures must be adopted as in younger individuals. Fresh dust-free air and sunshine are of the greatest importance. The dietary requires careful study as the senile digestive organs act more slowly and are often perverted, and the diet applicable to the young may be wholly inappropriate for the aged. The milk and egg diet useful in younger individuals soon becomes objectionable, and the amount of fluid that must be taken imposes excessive work upon the circulatory apparatus and kidneys. The food should be easily digestible, as concentrated as possible, and leave little waste. This excludes food containing a large amount of cellulose, fruits, jellies, foods fried in fat, and food that must be swallowed in lumps. The cereals

and leguminous vegetables should form the bulk of the food. Meats, if given at all, should be well cooked, and if the teeth are too defective for mastication it should be omitted altogether or meat juice should be substituted. Persons accustomed to alcoholic drinks need not be deprived of them, but the distilled liquors and wines should be diluted. If the sputum becomes blood-streaked alcoholics must be omitted.

Sanitarium and health resort treatment is rarely required. High altitudes are dangerous for aged tuberculosis cases and a moist atmosphere is liable to raise the blood pressure and increase the tendency to hemorrhage.

Medicinal treatment is indicated only for the relief of distressing symptoms and for complications. Turpentine, guaiacol and eucalyptol may be used by inhalation if there is an irritating cough with scanty, tenaceous secretion, or if the mucus has a fetid odor. Guaiacol and creosote should not be given by the stomach as they soon produce anorexia and may cause gastritis. They are to be used by the inhalation method which is more direct and more effectual. Treatment by tuberculin has been often quite successful. If the cough is very distressing and prevents sleep dionin or heroin in 1/12- to 1/8-grain doses can be given. Dyspnea is seldom severe enough to require treatment unless occurring after excessive exercise. If it becomes necessary to give relief for this then the inhalation of 3 minims of amyl nitrite will give immediate results. For prolonged treatment the nitrite of soda in 1/6- to 1/2-grain doses three times a day should be used. Intestinal disorders are always due to local conditions, generally to tubercular ulcers, sometimes to excessive or improper food, or to drugs. Constipation may be due to atonicity of the intestines or to drug action. If there is no obvious cause a mild laxative, such as castor oil, should be given and all food and drugs withheld for a day. If diarrhea continues there is a persisting local cause, probably an ulcer. In this case intestinal antiseptics, as salol, the sulphocarbolates, urotropin, etc., should be used in combination with a mild astringent as bismuth subnitrate. The food should be predigested, or partly digested, or food preparations which leave little or no waste should be used instead. Acids and acidulated drinks should be omitted, but buttermilk may be taken.

Miliary or **acute general tuberculosis** is rare in the aged and when it does occur it is invariably fatal. The disease is always

secondary to a local infection, but this may have been so mild as to have escaped detection. Miliary tuberculosis presents the constitutional symptoms of toxemia, but the local symptoms, arising from the organs or tissues containing the tuberculous lesions, predominate. These local symptoms are usually severe results of the primary infection. In some cases the constitutional symptoms, chills, fever, headache, prostration, etc., are severe from the onset, while the local manifestations are mild, and the patient passes rapidly into a typhoid state. There may be then a muttering delirium, insomnia, subsultus tendinum, picking at the bed clothes, dyspnea, cyanosis, a weak, rapid pulse but a low temperature, involuntary discharge of urine and feces and rapid emaciation. Death in these cases occurs during the first or second week. In the aged the disease usually assumes a toxemic pulmonary type in which the pulmonary symptoms predominate resembling acute lobar pneumonia or a capillary bronchitis. The primary lesion in these cases is a fibroid phthisis which is often not recognized until the acute miliary tuberculosis has appeared. In rare cases the constitutional symptoms are mild, fever is absent, but there are symptoms of a secondary infection in other tissues, generally in the brain. In other cases the mild symptoms of a fibroid phthisis suddenly become severe, there is cyanosis, dyspnea, a hacking, painful cough, blood-streaked mucopurulent expectoration, rapid emaciation and loss of strength and finally, death from exhaustion. In other forms of miliary tuberculosis, the cerebral symptoms predominate and the disease appears as a meningitis. In others again there is a toxemic pleuritic form and Ortner describes a marantic form which occurs in the aged. The predominating symptoms of this are a rapid loss of strength and waste of tissue without marked local or other constitutional manifestations. In rare cases there is an apparent improvement lasting for a day or two, possibly longer, with a fatal relapse. In determining the diagnosis the finding of the pathogenic bacillus is the most important factor. Diseases which resemble it clinically in its onset are influenza, sepsis, typhoid fever, pneumonia, bronchitis, meningitis, and actinomycosis. Where there is a history of tuberculosis the diagnosis is clear. In most cases a bacteriological test is necessary to determine the character of the infection, but an early diagnosis of miliary tuberculosis can often be made by the tuberculin test.

Treatment.—There is nothing we can do except to relieve the symptoms and our efforts in this direction often fail. Narcotics and hypnotics can be used to relieve pain, insomnia and occasional delirium, and expectorants or stimulating inhalations to increase and liquefy scanty and tenaceous expectoration. There is nothing known that will retard the rapid emaciation and loss of strength. Stimulants are but of momentary utility, the exhaustion keeping pace with the emaciation. In diseases such as this, which in the present state of knowledge are classed as absolutely fatal, we are justified in making any experiment, however irrational it may appear, to prolong life. (This would even justify the implantation of the germs of an antagonistic disease if the latter offers a possibility of longer life than the original disease.)

Bone tuberculosis is occasionally found in the aged, being carried over from earlier life either as the continuation of a slowly progressive tubercular process or as a recrudescence of the process after years of apparent cure. In the latter case the disease is usually active, rapid in its progress, there are many foci in the bones and organs, emaciation and debility proceed rapidly and the patient succumbs in a few weeks or months. In the slowly progressive form the disease may appear in the bony structure secondary to visceral tuberculosis or it may be the continuation of an original bone tuberculosis, usually a Pott's disease. The secondary disease usually attacks the foot, knee, hip, wrist or elbow. Tuberculosis in these locations is frequently mistaken for rheumatic arthritis or chronic rheumatism, occasionally for syphilis, osteomyelitis, rarely for sarcoma. The diagnosis should not be difficult if we remember that tuberculosis and syphilis give histories pointing to these diseases, that rheumatic arthritis produces characteristic deformities, that the tubercular joint is always swollen, blanched and has painful points. The pain is, however, never as severe as in osteomyelitis. The history alone will usually suffice to determine the diagnosis, but to clear up all doubts the tuberculin test may be necessary.

Pott's disease is usually carried over from maturity. When originating in advanced age its progress is rapid, there is considerable pain, the normal spinal curve becomes altered, a distinct bend being found at the site of the bone lesion; occasionally there is abscess formation, and there is marked cachexia. A neural form of vertebral tuberculosis is described in which the

Femur, head and neck; beginning tuberculosis. *A*. Small area of caseation in epiphysis just under articular cartilage, called subchondral. *B, B*. Same in epiphyses at point of junction with shaft; intraosseous. *C*. Subperiosteal. (*After McArdle* (*redrawn*) "*Trans. Royal Acad. of Med. in Ireland*," *vol. vii*, 1889, *p.* 140.)

only symptoms are those of myelitis and neuritis. The diagnosis must be made by excluding the various forms of spinal sclerosis and other diseases giving localized spinal pain. Pic says radiography performs a great service in diagnosing this form of Pott's disease. There is also a latent form of vertebral tuberculosis which gives no marked symptoms, but the lesion is found after death.

The treatment of bone tuberculosis in the aged is the same as in younger individuals.

Relapsing fever does not occur in the United States and is infrequent in the aged even in countries where it is met with. According to Ortner it does not differ from the disease as it occurs in earlier life, except that it is more fatal in the old owing to the grave secondary complications, pulmonary disease, suppurative parotitis and cardiac exhaustion. Experimental treatment with an antispirochetic serum has been reported favorably.

Miliary fever and *Malta fever* do not occur in United States and are rare in the aged in countries where they are epidemic.

Cerebrospinal meningitis is rare in old age, only 1 per cent. of the cases reported in the last New York epidemic occurring in persons over 50 years of age. The disease presents some minor differences in symptomatology from the disease in younger individuals. It begins usually with slight chills, the temperature is but little higher than normal, and in some cases may be normal or subnormal. The pain in the lumbar regions appears early, is intense, and proceeds rapidly upward along the spine. The rigidity of the muscles of the neck usually appears on the first day and prevents forward and backward motion, but lateral motion may not be impaired for several days. Opisthotonus is rare. The cerebral symptoms are severe from the onset of the disease. These are intense headache, photophobia, unequal dilatation of the pupils, impairment of the motor oculi, perhaps strabismus, nystagmus or ptosis, sometimes acute oversensitiveness of hearing, but more often deafness. In some cases there is paralysis of the facial and trifacial nerves, and the senses of taste and of smell become blunted. The tendon reflexes are diminished and may be abolished. Dermographia and facial herpes are frequently present. Kernig's sign is always positive. Pulse and respiration are accelerated, weak and irregular and Cheyne-Stokes respiration sometimes occurs shortly before death. There is almost always a rapid

loss of weight. Remissions occasionally occur and during apparent convalescence the symptoms will suddenly return with increased violence; in most cases, however, the disease progresses rapidly, delirium and then coma follow and the patient dies. A case may be protracted for several weeks, death finally ensuing as a result of exhaustion, pulmonary edema, or other complication. Many cases die before it is possible to make a positive diagnosis. The presence of Kernig's sign points to meningeal disease and the prevalence of an epidemic is presumptive evidence of the existence of the disease if there is prostration, lumbar pain and rigidity of the muscles of the neck. The onset in the aged is almost always clearly marked and can generally be diagnosed without the necessity of making a lumbar puncture to determine the character of the germs present. The finding of the diplococcus of Weichselbaum in the cerebrospinal fluid or in the secretion of the nose makes the diagnosis certain. In the treatment of cerebrospinal meningitis good results have been obtained from the use of the Flexner antimeningitis serum. In the pandemic of 1904 to 1908, the mortality where it was used was less than 25 per cent., while where it was not used the mortality was 70 per cent. It was less effective in senile cases than in younger ones. Lumbar puncture with the withdrawal of from 20 to 40 c.c. of cerebrospinal fluid, replacing it by a like amount of normal saline solution, often relieves the symptoms temporarily. The procedure must be repeated every second day. Ortner combines the two methods of treatment, replacing the withdrawn fluid by the antimeningitis serum. The hot bath treatment and Bier's hyperemia treatment cannot be applied to the aged, owing to the impaired circulation. Local disinfection of the throat and nose by means of peroxide of hydrogen is beneficial as a prophylactic measure. The treatment of the symptoms is directed principally to relieve the cerebral manifestations. The most important of these measures is the application of cold water, not ice, to the head and neck. This must be used continuously during the disease. Other remedies for the relief of the insomnia, headache, neuralgias, constipation, etc., must be selected with due regard to the condition of the heart and blood-vessels. Veronal, morphine combined with atropin, the bromides and aspirin may be used.

Acute articular rheumatism is almost always a recrudescence of a former attack. The symptoms do not differ from the disease in earlier life. In some cases there is little fever or swelling of the joints, but there is the same pain and stiffness. Occasionally, fever precedes the acute attack, and this prodromal fever may be higher than the fever that usually accompanies the involvement of each new joint. In other cases there are marked cerebral symptoms, with hyperpyrexia showing a severe toxemia. These cases are grave, since they sometimes proceed to delirium, followed by coma and death. Acute endocarditis, myocarditis and pericarditis, which are frequent complications of acute articular rheumatism in earlier life, are rare in the aged. The only serious complication that occurs frequently is hypostatic congestion followed by pulmonary edema, and this can usually be avoided.

The only disease which may be mistaken for acute articular rheumatism is gout and the differentiation should present no difficulty if we remember that gout generally affects the small joints, that intense exacerbations generally occur at night, and that there may be gouty deposits and a clear history of gout.

The treatment of acute articular rheumatism in the aged is the same as in younger individuals. The aged require large doses of the salicylates, about 20 grains every four hours; and morphine may be given if the pain is severe. The iodides are worthless in this disease in the aged.

Erysipelas occurs frequently in persons of advanced years. Its favorite location is about the lower extremities, and it appears there more frequently than in maturity, but it may also occur about the buttocks, face, head, hands and other portions of the body. The prevalence of erysipelas about the lower extremities is explained by the frequency of excoriations, varicose ulcers, eczema, scratches and abrasions, following pruritus in that locality. When occurring about the buttocks it follows bed sores; when on the face it follows some slight lesion about the nose, eye, corner of the mouth or elsewhere. Erysipelas of the scalp is usually an extension of the disease from the face. The source of infection in every case is a local primary lesion which may have been so insignificant that it escaped notice. Lack of cleanliness is a contributing factor in most cases. Owing to the senile changes in the skin—atrophy of subcutaneous

tissue, partial obliteration of capillaries, and diminished surface circulation—the local symptoms are generally modified. The redness is not as intense as in maturity, there being less infiltration and swelling and little or no elevation of temperature in the affected part. It is frequently localized in a small area, and spreads slowly. In some cases there are patches of erythema joined by fine reddish lines. It rarely spreads by the lymph spaces, mostly by surface extension. Sensations of heat and pain are much slighter than in earlier life and but rarely is there any involvement of the local lymphatics. Vesicles, blebs, and pustules are seldom seen, but gangrene may occur in localities where the circulation is greatly impaired. The constitutional symptoms, like the local ones, are much milder in the aged than in earlier life. The onset may be abrupt, with a severe chill; more often there are slight chilly sensations followed by a rapid rise in temperature. The latter may be but little raised, rarely higher than 102°. The pulse and respiration are accelerated, but after defervescence of the eruption the pulse becomes slow and may drop to 50 per minute while the temperature may sink to 95°. Erysipelas of the face is often accompanied by bronchial catarrh; while erysipelas of the scalp is generally accompanied by cerebral symptoms such as intense headache, delirium, delusions, hallucinations, insomnia, etc. Albuminuria is generally present but other complications, which usually accompany infectious diseases, are rare. Relapses, however, may occur after an apparently complete recovery, and recrudescence months or years after the original disease had disappeared is not rare.

The treatment of erysipelas is prophylactic, curative, symptomatic and hygienic. The principal prophylactic measures are cleanliness; antiseptic treatment of all wounds, ulcers, excoriations and scratches and the quarantine of cases when they occur in institutions. The curative measures include serum therapy, which is still in an experimental stage, and measures to localize and diminish the eruption. For this purpose ichthyol is probably the most effective. It is brushed thickly over the affected area, covered with cotton, and allowed to remain until twenty-four hours after all local and constitutional symptoms have disappeared. This is superior to resorcin, guaiacol, nitrate of silver, or tincture of iron, the drugs that

were formerly employed for the purpose. Hot fomentations sometimes relieve local pain, but they do not improve the general condition. Carbolic acid, the lead, mercury, silver, and other metallic salts are contraindicated in the aged. For the relief of distressing symptoms, like headache, fever, insomnia, pain, etc., the usual remedies for such conditions are required. Cold applications, not ice, can be applied to the head if cerebral symptoms appear. For the fever the preferable drug is quinine or salicylate of soda, but none of the coal-tar preparations should be used. Veronal may be given for insomnia. If local pain is severe, hot applications of tincture of opium or a 5 per cent. cocaine ointment can be applied. Internal analgesics are seldom required. The hygienic regulations require only a nutritious diet with little carbohydrate and no hydrocarbons; care of the bowels and kidneys, drinking large quantities of alkaline water if the renal secretion is deficient in quantity, and observance of the ordinary rules of health.

SEPSIS

Sepsis is used here to include *septicemia* and *pyemia*. Much confusion has arisen through different interpretations given to these and their allied terms toxemia, bacteremia and septico-pyemia. The last of these terms is superfluous, as every pyemia is septic and produces symptoms of septicemia. The term toxemia is usually applied to a condition in which bacterial toxins exist in the blood and bacteremia is applied to a condition in which the bacteria themselves are present in the circulation. Septicemia is applied in its broadest sense to the disease produced by toxemia or bacteremia, while in its narrowest sense it is restricted to the disease caused by pus-forming germs or their toxins before secondary foci of suppuration have developed. A localized pus formation in which the local symptoms predominate receives a local appellation as pyelitis, purulent pleurisy, abscess of the lung, etc. When the pus is carried in the blood and deposited in various localities, and the systemic symptoms predominate, the disease is pyemia. In furunculosis, for example, there are many local pus deposits but the systemic symptoms are mild. Pathologically it is a pyenia, clinically it is not. Ortner rejects the term pyemia and speak of it as

metastatic sepsis, reserving the term true sepsis for septicemia. Other writers take different views.

Sepsis like most other infectious diseases is infrequent in the aged and when it does occur it runs an atypical course. The usual channels of infection in the aged are surface lesions such as chronic ulcers, eczema, erosions, scratches, bed sores, etc., or the bladder infected by catheterization. Less frequently the channel of infection is the lower bowel, the nose, mouth, respiratory or digestive tract. In some cases the source of infection is in the gall-bladder or in the ducts, rarely in the serous membranes.

The symptoms of septic infection in the aged differ somewhat from those of earlier life, the most pronounced differences being in the lower temperature and more frequent cerebral symptoms. Even in a grave form of sepsis the temperature rarely exceeds 103°. When due to the streptococcus the temperature is irregular, sometimes it is continuous for days, sometimes remittent or intermittent. The bacillus coli produces an irregular temperature with frequent slight chills, the temperature rising after each chill, then dropping until the next chill occurs. In some cases these chills come on at quite regular intervals and there is then a fairly regular rise and fall of temperature. The same condition may also be due to staphylococcic infection and this has been considered as distinctive of pyemia. In many senile cases of sepsis the temperature does not rise above 98.5°. There is rapid heart action and rapid respiration in spite of the low temperature, frequently dyspnea, weak pulse and always some cerebral disturbance. This is generally evidenced by severe headache and insomnia, sometimes by more profound disturbances such as photophobia, delirium and coma, involuntary discharge of feces and urine, and great prostration. The blood changes are generally similar to those of maturity but in debilitated patients, especially if the infection is severe, leucopenia may exist from the onset of the disease or may appear after a short leucocytosis. The spleen is rarely found enlarged, but this is only relative as the senile spleen is normally diminished in size. Constipation is usual owing to intestinal paresis. The cutaneous manifestations frequently found in earlier life are rare, except a temporary herpes, erythema or a roseola. The most serious and most

frequent complication of sepsis is septic endocarditis. Apart from the danger to the heart itself, septic endocarditis gives rise to emboli which may be carried to any of the tissues, producing infarcts, abscesses, and hemorrhages. Abscess formation occurs most frequently with staphylococcic infection. The abscesses are usually small and scattered throughout the tissues so that their exact location cannot be determined. Occasionally an abscess is limited to a single organ such as the lung, or occurs in a single tissue such as a joint, or will burrow through adjoining tissue and form a pocket at some distance from the original site. Occasionally a local septic inflammation with pus formation will destroy the enclosing tissue and the pus pouring into one of the large cavities causes a septic peritonitis, pleurisy, cellulitis, etc.

Sepsis in the aged is usually fatal. It will not occur unless the resistance of the body is lowered and that alone implies lowered vitality. In the cases where surgical measures can be taken to empty pus depots, recovery may follow. Where such depots cannot be reached or where there is a virulent, non-suppurative septic infection a fatal issue may be expected. How far serum therapy will modify the prognosis is uncertain. In this, as in all other infectious diseases of the aged, the profound physical depression is of graver import than the local action of the germs. The prostration is often far greater than the extent or virulence of the infection would account for and persists after the disease germs have disappeared. Serum therapy of the future, if effectual, will destroy further germ activity and shorten the length of exposure of tissue to the deleterious influences of the germs, thereby removing complicating factors.

The invasion of most infectious diseases in the aged gives a very similar symptomatology and only bacteriological examination can determine an early diagnosis. In a mild pyogenic infection there will be found a leucocytosis. This excludes typhoid fever, malaria, acute tuberculosis, influenza, and measles. In a virulent infection there is leucopenia with relative increase of polynuclear leucocytes, while in the leucopenia of typhoid fever the lymphocytes are relatively increased. The Widal test and diazo reaction will differentiate typhoid fever. In the latter disease the initial prostration is more pro-

found, the abdominal symptoms appear early and there are rarely chills, herpes, or the rapid pulse and respiration found in sepsis. When typhoid fever and sepsis are present at the same time the typhoid symptoms will completely mask the symptoms of sepsis unless abscesses form. The early differential diagnosis between sepsis and miliary tuberculosis may sometimes be made by the history either of a fibroid phthisis or other form of tuberculosis, or else of a surface lesion. Cyanosis and cough occur early in acute tuberculosis; late, if at all, in sepsis. The absence of catarrhal symptoms will exclude influenza, and malaria can generally be excluded by the history and the condition of the patient after the attack.

The present-day treatment of sepsis is by serum therapy. The frequent failures where serums and vaccines are used arise from using a single strain of polyvalent vaccine in cases in which several forms of bacteria are active. Success by this method of treatment can be achieved only when we know the kind of bacteria we are dealing with and for that reason a bacteriological examination should be made before we select the serum or vaccine. A combined vaccine can be used if several forms of bacteria are found. In advanced arteriosclerosis and weak heart the sera and vaccines are contraindicated. (French physicians recommend as a curative measure the subcutaneous injection of colloidal metals such as electrargol, electroplatinol, etc. Their therapeutic value is uncertain, however.)

Other measures in sepsis are either surgical or measures for the relief of symptoms.

In the treatment of symptoms we must bear in mind the senile changes. We must not use powerful vasoconstrictors like digitalis, nor cardiac depressants like the coal tar products and chloral, nor drugs which inhibit peristalsis like belladonna and opium. Cold baths may produce a fatal shock and ice may destroy the surface circulation. The safest drug for reducing the temperature is quinine. Its action, however, is slow and it frequently causes gastric irritation. Heart tonics are required from the onset of the disease. The most available is caffein or coffee. In threatened heart failure we can use camphor, ether, or strychnine hypodermically. For the cerebral symptoms cold applications to the head,

and the bromides internally, and for insomnia we can use veronal or urethane.

Care should be taken to secure free bowel and kidney action. Any of the peristaltic stimulants as aloin, cascara, castor oil, etc., can be employed to prevent constipation, and for the kidneys nothing will take the place of water. It can be taken in small quantities in short intervals, never in large quantities at a time, or it may be given by large rectal enemata slowly delivered several times a day. Where surgically accessible pus depots exist, these should be cleaned out. When death without operation appears inevitable the most desperate surgical measures may sometimes succeed.

Gonorrheal infection is rare in the aged as they expose themselves less and there is apparently less susceptibility to the disease. Various hospital and dispensary statistics place the number of cases of gonorrhea between fifty-one and sixty years of age at little over 1 per cent. of the whole number of cases seen and above sixty years of age at a small fraction of 1 per cent. Gonorrhea in the aged female is extremely rare. The symptoms do not differ from those of earlier life. The disease is usually milder, but less amenable to treatment and is often followed by a postgonorrheal urethritis, but seldom by a prostatitis or stricture. Other complications are rare. An infectious non-gonorrheal discharge simulating gonorrhea is sometimes found in those who fail to observe antiseptic precautions when using the catheter. The diagnosis in such a case rests upon the bacteriological findings. Prostatorrhea, spermatorrhea and simple urethritis may occur in the aged and give rise to the suspicion that a gonorrhea exists. These all require a microscopic examination to determine their character. When a stricture is suspected a sound must be used. The stricture may be simulated by urethral spasm and compression of the urethra by a hypertrophied prostate. The former disappears after an injection of a 2 per cent. solution of cocaine in warm water. The latter gives other symptoms pointing to an enlarged prostate, while the history of gonorrheal infection is absent. A postgonorrheal stricture, however, may exist at the same time with a hypertrophied prostate.

The treatment of gonorrhea in the aged is the same as in young individuals. If there is a stricture, slow dilatation by

means of sounds is better than the more rapid divulsion or incision methods.

General infection, gonorrheal toxemia, gonorrheal arthritis, gonorrheal endocarditis and gonorrheal neuritis, etc., are extremely rare in the aged. They must be considered among the possibilities, however, where there is a toxemia, arthritis, endocarditis, neuritis, etc., of unknown origin, but unless the specific organism is isolated in the blood or synovial fluid, we cannot make a diagnosis of systemic gonorrheal infection. If the diagnosis has been confirmed, we can use the gonorrheal vaccine, subcutaneously, as a curative measure. The septic and endocardial forms are usually fatal.

SYPHILIS

Syphilis is seldom acquired in advanced age. Persons having congenital syphilis rarely reach old age and old people do not expose themselves to the danger of infection as often as younger persons. Fournier reports of 10,000 cases, 207 between the ages of 51 and 61 and 40 between the ages of 61 and 71. Tertiary syphilis occurs more frequently, hospital records showing from 8 to 13 per cent. over the age of 50; of these, 1 to 3 per cent. were between 60 and 70, and a small fraction of 1 per cent. over 70. In many senile cases the disease is acquired accidentally in old age and does not differ then materially from the disease in younger individuals. Sometimes the period of incubation is prolonged and the initial lesion persists longer. The sore is often larger and deeper than in maturity, it looks raw and in some cases it becomes gangrenous. The disease on the whole is usually more severe than in younger persons and secondary lesions may appear before the primary chancre has disappeared. The lymphatics become very slowly and at times not severely involved, but the cutaneous and nervous manifestations are more pronounced than in earlier life. A diffuse papulopustular syphilide is common. Syphilitic iritis is an early manifestation of the second stage. There is no sharp dividing line between the second and the third stages, the disease in the aged usually progressing without intermission. The pustules become ulcers, gummata form, the internal viscera become affected early, usually by the production of syphilitic ulcers, the syphilitic cachexia is pronounced and often leads to fatal exhaustion. Quinquard found a constant de-

crease of the red blood cells, hemoglobin and of the albuminoids of the serum, the red cells numbering as low as 2,000,000. The central nervous system is often profoundly affected, and there is usually mental depression, irritability, headache, sometimes insomnia and vertigo.

In some cases the invasion of the secondary stage is like the invasion of an eruptive fever, with chills, fever, prostration, headaches, etc., and only the history, the Wasserman test, or the finding of the spirocheta will determine the diagnosis of syphilis. While French physicians generally ascribe greater virulence to the disease acquired in old age, the opposite view is held by German physicians. American physicians who see many syphilitic cases confirm the French view. The disease, however, does occasionally appear in a mild form in the aged, the initial lesion is small, the secondary symptoms begin with a slight roseolar rash, the mucous membranes are not affected, there are no pains in the bones or joints, and tertiary symptoms do not appear. Fournier described, under the name *La Cachaxie adynamique*, a rare form of syphilis in the aged. In this form there are pronounced constitutional symptoms with little or no local ones, except the initial chancre. There are mental and physical depression, anemia, anorexia, somnolence, progressive emaciation and exhaustion. In severe cases the fatal issue is reached in a few months, more protracted cases may last two or three years, death being due to exhaustion or pulmonary edema. Most cases of syphilis in the aged resemble the third stage of a syphilis which had been apparently cured years before. Fournier reported a case in which the tertiary lesions appeared fifty-five years after the infection. In these delayed cases of tertiary syphilis the disease is generally mild and is confined to one locality or tissue such as the skin, mucous membrane, bone, etc. If a viscus is affected it is generally by a chronic syphilitic ulcer, which gives little pain and no other clear symptom.

The finding of the spirochetæ in the blood establishes the diagnosis with positiveness. If these are absent we can use the Wassermann reaction. A positive reaction means a positive diagnosis, but we may get a negative reaction though the disease be present. If both of these methods fail we have still the history of exposure and initial lesion. This initial chancre is present in every case, but its site is not necessarily confined to the genital organs. Aside from the rare cases of *syphilis inson-*

tium or accidentally acquired syphilis, the frequency of sexual perversions in the aged, in whom the potentia coeundi has diminished, gives rise to unusual locations of infection. The unreliability of such perverts makes the history, as obtained from them, unreliable. A chancre should, however, give no difficulty in diagnosis and it is only after secondary or tertiary lesions arise, and history, as well as bacteriological examination, and Wassermann reaction are all negative that there can be any question as to the correctness of the diagnosis. During the second stage the brownish spots, mucous patches, atrophy of the glands at the base of the tongue and enlargement of lymphatic glands form a pathognomonic symptom-complex. Should there be any question of diagnosis in the tertiary stage we will usually be able to get a history of the symptoms of the secondary stage even if an initial chancre is denied. In questioning the aged where there is a suspicion of syphilis, more truthful replies will be obtained if we enquire about the secondary lesions without explaining the purpose of the questions, for they will generally deny having had a venereal disease. The tertiary symptoms in the aged are frequently misleading. Syphilitic eruptions and sores do not itch, but the aged often suffer from a pruritus independent of syphilis and the cutaneous lesions may then itch. Sclerotic degenerations are more often due to senile changes than to syphilis and the same applies to anemia, emaciation, albuminuria, constipation, valvular diseases, all of them conditions which may be due to syphilis, yet which are found normally in the aged. They may also occur in tuberculosis and the differentiation between a tuberculous ulcer and a syphilitic ulcer will sometimes be impossible without a bacteriological examination, the Wassermann test or the tuberculin test. If all these diagnostic methods fail we must use the red or yellow iodide of mercury and observe the result, an improvement occurring in syphilis but not in tuberculosis.

The treatment of syphilis in the senescent is the same as in maturity. The older method of treatment with mercury and the iodides is still the most reliable one and with slight modifications can be applied to the aged. The mercury should be used by injection or inunction rather than by mouth and the insoluble salts are preferable to the soluble ones for internal administration. A salicylarsenate of mercury, is highly ex-

tolled by French physicians. Sodium cacodylate is much used in this country. The old Donovan's solution of arsenious and mercuric iodides, containing the three antisyphilitic remedies, may be tried. Apart from the uncertainty of the action of the newer arsenical remedies such as salvarsan and neosalvarsan upon the senile organism, the aged will rarely permit a repetition of their intramuscular injection owing to the pain, but an intravenous injection is free from this objection. For local manifestations of the disease, such as syphilitic ulcers including the primary lesion, bismuth subnitrate and calomel in equal parts may be used as a dusting powder. If the chancre has become phagedenic it should be cocainized and touched with a drop of acid nitrate of mercury or of pure nitric acid. For the enlarged glands the oleate of mercury in a 5 per cent. ointment should be used. The scarlet red ointment is said to cure localized lesions.

GENERAL ANEMIA

The old classification of anemias into primary or idiopathic and secondary or symptomatic anemia is convenient rather than correct, as it is based upon etiological factors, the cause of some being unknown. These are progressive pernicious anemia and chlorosis, to which some authors add leukemia and pseudo-leukemia. It is not at all certain that pernicious anemia and chlorosis are not due to bacterial or toxic influences and hence are symptomatic anemias similar to the anemia of cancer or malaria. Anemia includes many forms of blood changes. In oligemia the entire quantity of blood is diminished. In hydremia the proportion of water is increased with the consequent proportionate diminution of the other elements. In oligocythemia the proportion of red cells is diminished. In hemoglobinemia the percentage of hemoglobin in the cells is reduced. In leukemia the white cells are increased. In some forms of anemia the albumin content is diminished, in other forms there is a change in the character of the cells. All forms of anemia except chlorosis are found in the aged.

Oligemia.—A diminution in the total quantity of blood in the aged was noted by Geist. This is due to the degeneration of the hemapoietic system, to contracted blood-vessels, obliterated capillaries and diminished thirst. The composition of the

blood is not altered. This oligemia vera is marked in poorly nourished individuals in whom there exists atrophy of all the tissues, though the individual remains in fairly good health. This being a physiological condition in old age, nothing can or need be done for it. Inorganic salts of iron will not increase the hemoglobin percentage and the increased ingestion of food and drink will not improve the degenerated spleen or bone marrow nor the capacity of the blood-vessels.

In traumatic oligemia, the diminished amount of blood is occasioned by hemorrhage. It may be a slow persistent bleeding as from hemorrhoids or cancer, or a sudden severe hemorrhage as when a vessel is cut. In sudden hemorrhage there is intense thirst, a physiological provision for replacing the lost fluid. The blood-forming tissues, however, are degenerated in the aged and repair proceeds slowly if at all. While in maturity the injection of normal saline solution will generally prevent the collapse following profuse hemorrhage, and will sustain the patient until the spleen and marrow have replaced the lost cells, the aged frequently succumb, unless transfusion is performed. In slow persistent hemorrhage the cause must be removed if possible and nutritious food supplied. Iron medication in the aged is generally worthless. The inorganic forms of iron are not readily assimilated and the organic preparations do not increase the hemoglobin percentage nor the number of red cells. Iron-holding foods such as green vegetables, salads, spinach, cabbage, young beans, peas and lentils are recommended. Red bone marrow from the long bones of young animals will increase the number of red cells where they are deficient, but the remedy soon becomes objectionable to the patient.

Hydremia.—This is the usual condition of the blood when the cells are damaged by bacterial or toxic influences. It may also occur when there has been slow inanition with a large ingestion of liquids.

Hydremia gives rise to local edemas, miliary hemorrhages, and to irregular heart action. The treatment depends primarily upon the cause. For the removal of the excessive amount of fluid, diuretics and diaphoretics must be used, the selection of the drug depending upon the condition of the heart and kidneys. The saline cathartics, in concentrated solution, are often effective in this condition.

Albumin Deficiency.—Grawitz has shown that the anemia of inanition is not due to a deficiency of iron but to a general deficiency of albumin with consequent deficiency of albumin in the plasma. As a result of this impairment of the plasma the red cells degenerate. Whatever will cause disturbance in the assimilation of albumin will produce this form of anemia, and it is therefore found most frequently in gastric atony and dyspepsia. The treatment depends upon the cause.

Hemoglobinemia.—The agents which destroy the red cells first release the hemoglobin, which is then carried by the serum to be converted in the liver and eliminated by the kidneys as hemoglobin or methemoglobin. As the hemoglobin is released before the destruction of the cell we find the hemoglobin percentage proportionately lower than the cell count. In pernicious anemia the red cells though greatly diminished in number are generally very large and may contain then as much hemoglobin as the healthy cells, consequently we get a high color index in spite of a low blood count. In those cases in which there is a low hemoglobin percentage sterile iron by hypodermic in combination with arsenic may be tried or it may also be given in the form of hemoglobin and arsenic while manganese may be added. In every case, however, it is necessary to reach the cause of the anemia and remove that before we can expect permanent results.

Oligocythemia.—Diminution of the number of red cells is always found in anemia. In oligemia the proportion is maintained, but in every other form the proportion is reduced, the lowest number recorded being 143,000 per cubic millimeter, and that was in a case of progressive pernicious anemia. In anemia the red cells degenerate before they are destroyed and they present various abnormalities in size, shape, staining qualities, hemoglobin percentage, and the presence of nuclei. The principal causes for anemia in the aged are malignant disease, malaria and other infections, chronic suppuration, chronic dysentery, chronic nephritis, cirrhosis of the liver, metallic poisons, intestinal autointoxication and intestinal parasites. Pernicious anemia is believed to be due either to a specific micro-organism or to an autointoxication of gastric or intestinal origin.

The only idiopathic, primary form of anemia is oligemia. All other forms are secondary to another disease or part of a more

comprehensive pathological condition. It is rarely possible to make a diagnosis of the underlying condition from the count or character of the red cells alone, as all the various abnormalities may be found in any of the severe anemias. Only in pernicious anemia must we depend upon the blood count for our final diagnosis.

The most marked symptoms of anemia occur in profuse hemorrhage. These are pallor, vertigo and faintness, prostration, palpitation, blanched mucous membranes, and cold perspiration. In the chronic anemias all these symptoms, excepting the pallor, are slight or absent. Most of the diseases associated with anemia, occurring in the aged, give distinctive symptoms apart from the anemia, and only progressive pernicious anemia need be considered as a distinct disease in which the blood changes present the main diagnostic factor. The hook worm disease, uncinariasis, resembles pernicious anemia in its symptoms, but it presents as a diagnostic sign the eggs or worms in the feces.

Pernicious Anemia; Etiology.—The cause of pernicious anemia is unknown. Its course would indicate the activity of bacteria or of a bacterial toxin though no specific germ giving the distinctive symptoms or producing the marked changes in the red cells has been found. In many cases there is undoubted intestinal autointoxication; in some cases lead poisoning and carbon dioxide poisoning have produced similar symptoms. Persistent bleeding from the gastrointestinal tract may give symptoms of a rapid progressive anemia. Other possible causes that have been suggested are degeneration of the marrow whereby regeneration of the blood is interfered with; atrophy of the stomach, this condition being frequently found after death; septic infection, septic lesions generally existing in the gastrointestinal tract, embryonic cells of another species of animal, etc.

It seems probable that many substances which find their way into the blood have a deleterious influence upon the cells and cause rapid impairment and destruction. However, only a single etiological factor in the nature of a bacterial toxin can produce the profound cell changes found in a typical case of pernicious anemia. Many cases occurring in the aged give the ordinary symptoms of pernicious anemia and show a low red cell count without the large number of megaloblasts or the extreme poikilocytosis found in the typical form of pernicious anemia.

In these cases the bone marrow is found degenerated and the anemia is evidently due to impaired hemapoietic activity. In other cases the regulation of the diet by the withdrawal of all forms of animal albumin produces a rapid improvement. In some cases, however, there is a rapid poikilocytosis with a large number of megaloblasts, and the red cells are diminished to 2,000,000 or less per cubic millimeter, and none of the measures employed to improve the digestion, eliminate poisons, control internal bleeding or overcome septic infection have the slightest effect upon the disease.

Symptoms.—The most pronounced symptom of a typical case is a peculiar pallor, not sallowness as in cancer, but a waxy yellowish color. The mucous membranes are blanched and there is progressive bodily weakness without emaciation. The muscles become flabby, and slight exertion causes dyspnea and palpitation of the heart, with fatigue from which recuperation is slow. The appetite is usually lost and in most cases there are gastric and intestinal disturbances. Heart action becomes weaker and more rapid, systolic murmurs are heard over the mitral and aortic valves and blowing anemic murmurs over the aorta and sometimes over the carotids.

Retinal hemorrhage and purpura are frequent and occasionally the symptoms of miliary hemorrhage in the brain appear. In those cases in which the absorption of the products of intestinal putrefaction is supposed to be the cause, the urine contains a large amount of indican and small amounts of cadaverin, and other substances derived from intestinal decomposition.

The blood in pernicious anemia is profoundly altered. The red blood cells are greatly diminished while the hemoglobin percentage is not proportionately reduced. The cells are distorted in shape, there are many megaloblasts and a few normoblasts while platelets are increased. The leucocyte count is diminished.

As a result of the faulty nutrition of the tissues through the impairment of the blood, fatty degenerations occur most markedly in the heart and involuntary muscles. The diseases liable to be mistaken for pernicious anemia are Grawitz' cachexia and cancer. Grawitz' cachexia gives similar symptoms, but shows no blood changes. In some forms of cancer the only early symptom is the cachexia. This is associated with emaciation, the

pallor is a sallowness, there is leucocytosis and there is never the great reduction in number or the profound changes in the character of the red cells that we find in pernicious anemia. Retinal hemorrhage is frequent in the latter disease, extremely rare in cancer.

Pernicious anemia is a fatal disease though there are occasional remissions in the symptoms and occasional improvement in the character of the blood. Cases due to gastrointestinal disturbance are occasionally cured but it is doubtful if these were cases of true pernicious anemia.

Treatment.—If an underlying cause can be found, that cause must be removed if possible. Where there has been absorption of the products of intestinal decomposition, as evidenced by the indican percentage in the urine, intestinal antiseptics and the exclusion of animal albumin and other purin-forming foods are necessary. Gastric digestion should be stimulated by lavage, pepsin and fruit acids or hydrochloric acid, and intestinal activity should be increased by the administration of pancreatin and the bile salts. The sulphocarbolates are the preferable antiseptics in these cases.

Little can be done in cases due to persistent internal hemorrhage. Adrenalin solution will frequently stop the bleeding but is dangerous in old age and may cause apoplexy. The lime salts increase the viscosity and coagulability of the blood and may stop internal hemorrhage. In many cases the bleeding comes from a cancer and surgical measures may be indicated.

A pronounced oligocythemia without marked poikilocytosis points to degeneration of the blood-forming tissues. In these cases red bone marrow can be given, with hemoglobin, arsenic and manganese. If these measures fail when given internally they should be given hypodermically.

LEUKEMIA

Leukemia presents no marked difference from the same disease of maturity. Both the myelogenous and the lymphatic types occur in the acute and chronic forms. The acute form, which is generally of the lymphatic type, is very rare in the old, and is usually fatal in from one to four weeks. It resembles in its course a malignant acute infectious disease, beginning with

high fever, followed by rapid enlargement of the spleen and usually enlargement of the lymphatics of the neck, axilla, inguinal and other regions, hemorrhages from mucous surfaces, purpura, etc. The chronic form may exist as a slowly progressive cachexia for months before its nature is suspected. In some cases, glandular enlargement or abdominal distention is first noticed, in other cases bleeding from the gums or other hemorrhages first attract the attention of the patient. There are numerous vague symptoms such as gastric and intestinal disturbances, nervous symptoms, headache, vertigo, irritability, or a general malaise with a feeling of being very ill indeed without being able to refer the sickness to any one organ or tissue. In some cases, especially in the aged, the symptoms point in many different directions and it is impossible to make a diagnosis until the blood is examined.

The blood changes in leukemia are distinctive and a single glance through the microscope will suffice to determine the diagnosis. The leucocytes in myelogenous leukemia are increased from ten to two or three hundred times the normal number and many abnormal types appear.

In the lymphatic type the leucocytosis is not as great, but the lymphocytes form from 75 to 99 per cent. of the whole number of white cells. In no other disease is the leucocytosis as high, or are so many abnormal cells found. A lymphocytosis occurs in whooping cough, but here the clinical symptoms are distinctive. It also occurs in the rare diseases, myeloma and chloroma or green cancer.

The treatment of leukemia is unsatisfactory. The acute form is generally fatal in a few weeks. The chronic form presents occasional remissions with improvement under treatment, but relapses occur frequently. The most effectual treatment is by means of the X-ray and this has given even better results in the aged than in younger individuals. Of drugs, benzol has been used of late with remarkable success, and arsenic has had a proven beneficial effect. The latter is given in the form of Fowler's solution, beginning with 1 minim three times a day, and gradually increasing the dose a minim a day until the physiological effects appear, when its use must be discontinued for a few days, after which the maximum dose is given continuously until the cumulative effects appear again. Quinine and

iron, useful in younger individuals, have little or no effect in the aged.

PSEUDOLEUKEMIC DISEASES

Under pseudoleukemic diseases are included two diametrically opposite types, diseases resembling leukemia clinically but without the leucocyte changes and diseases having the leucocyte changes but not the symptoms of leukemia. Multiple lymphoma and splenomegaly belong to the first type, myeloma and chloroma to the second type. Lymphoma is seen occasionally in the aged, the others are rare and when occurring they do not differ from the same diseases of earlier life.

Lymphoma presents the clinical picture of a chronic leukemia in which the enlargement of the lymphatics is most marked. There is a slow progressive cachexia, the spleen is enlarged and hemorrhages from the mucous membranes as well as purpura are of frequent occurrence. The glands of the neck, axilla and inguinal region are most frequently affected. They enlarge but remain in their capsule and do not break down or ulcerate. Numerous other symptoms referable to the digestive, nervous and circulatory systems may appear. Fever points to infection. In making a diagnosis of this form of pseudoleukemia it is necessary to exclude leukemia and tubercular and syphilitic adenitis. The absence of leucocytosis and of abnormal cells will exclude leukemia. The respective serum tests may be required to eliminate tuberculosis and syphilis unless we can get a clear history of either. The disease may run a rapid course, or it may be slowly progressive, lasting for years before the cachexia causes fatal exhaustion. The treatment is mainly symptomatic, although arsenic and the X-ray have sometimes a beneficial effect, which is, however, not lasting.

RHINITIS

Acute rhinitis has the same etiological factors producing the same condition in earlier life, and the course of the disease is similar. Owing to the atrophic condition of the nasal mucous membrane, the local irritation is milder, there is less hyperemia and not so much mucous secretion, but a greater tendency to

involvement of the nasopharynx and conjunctivæ. Local treatment is rarely required unless the secretion becomes mucopurulent, when mild, non-irritating antiseptics like boracic acid, thymol, and aristol may be employed by insufflation, or the simple alkaline antiseptic lotions used as a douche. An elevation of temperature with aching limbs, headache, labial herpes, etc., indicates a bacterial infection. This is rarely severe and requires only rest, warmth, and small doses of quinine or aspirin, or a combination of quinine and Dover's powder, giving 5 grains of each twice daily.

Chronic rhinitis occurs frequently in the aged, but the symptoms are usually so mild that no attention is paid to it. It occurs in persons who have frequent attacks of acute rhinitis or who are constantly exposed to irritating dust or vapors or rapid changes in temperature. When following repeated acute attacks, it begins as an atrophic rhinitis, the last attacks of the acute disease having left the mucous membrane dry and thin. When due to constant irritation it begins as a hypertrophic rhinitis, with swollen mucous membrane and increased mucous discharge. The discharge becomes thicker and finally forms crusts, while the membrane underneath becomes thin and anemic. The crusts are irritating and cause the patient to remove them, thus leaving the underlying sensitive membrane exposed to further irritation. In many cases this leads to ulcerations which may extend to the bone and cause necrosis. There in a thin fetid discharge from the ulcerated membrane, the fetor becoming worse when necrosis of the bone occurs. This fetid coryza or *ozena* is usually ascribed to tuberculosis, but in the aged the ulceration and subsequent necrosis of the turbinated bones are generally due to repeated irritation by the patient's finger nails. Owing to the loss of the sense of smell the patient does not perceive the offensive odor, and this condition may persist for years before the injury to the bone will cause him to seek relief. There is generally marked erosion of the bones and the nasal cavity is enlarged, the nostrils being dilated by repeated stretching with the finger.

Atrophic rhinitis can be cured at an early stage by local medication. The nasal cavity should be thoroughly cleansed with an alkaline antiseptic solution, after which anhydrous lanoline should be applied, the patient drawing it up as far as possible.

This should be done several times a day and continued for a week. At the end of a week the treatment should be omitted for a day to see if mucus still crusts. If this occurs the treatment should be repeated. After necrosis of bone sets in surgical intervention becomes necessary.

DISEASES OF THE THROAT

Acute pharyngitis is rather infrequent as the mucous membrane is usually atrophied and it requires a powerful stimulus or irritant to cause acute inflammation. The symptoms are generally mild, there being little or no fever. The mucous membrane of the pharynx is not as red nor as swollen as in earlier life nor is deglutition greatly interfered with. The local symptoms rapidly subside upon spraying the throat with a 1–10,000 solution of adrenalin repeated every three hours. A temperature exceeding 100° in the aged points to an infection and the mucus should be examined for the spirillæ of Vincent's angina and for staphylococci and streptococci.

Vincent's angina is seldom found in the aged and its symptoms are much milder than in early life. The mucous membrane is covered with a yellowish or grayish exudate in which the pathogenic germs are found, there is a peculiar fetid odor to the breath and erosions and ulcerations of the mucous membrane of the mouth and throat occur. The constitutional symptoms are rarely severe. Frequent spraying with hydrogen peroxide followed by the application of tincture of iodine or a solution of iodoform in ether will generally cure this condition.

Chronic pharyngitis occurs frequently in the aged as a dry atrophic condition. It is due to prolonged irritation from substances inhaled or taken in food. A frequent cause is the inhalation of excessively dry warm air especially when sleeping with the mouth open in a room heated by hot air radiators. The symptoms consist of a sense of dryness in the throat that causes persistent thirst, and irritation produced by the small amount of tenaceous mucus that is secreted and remains adherent to the pharynx, causing hawking and coughing in an effort to dislodge it. In dealing with this form of pharyngitis we must first discover the cause. The atmosphere of the room can be kept moist by placing a vessel of water upon the radiators. To produce

local stimulation a 2 per cent. solution of menthol in a normal saline solution should be used as a spray. As chronic pharyngitis is usually associated with chronic rhinitis, the treatment suggested under Rhinitis should be combined with the treatment of the pharyngeal condition.

Acute tonsillitis is infrequent in the aged for the same reason that acute pharyngitis is rarely met with. The tonsil itself is usually atrophied and is rarely swollen. The treatment suggested for acute pharyngitis also applies to tonsillitis.

Retropharyngeal abscess is rare. When it does occur the etiological factors, symptoms and treatment are the same as in maturity. The same also applies to *peritonsillar abscess*. Other affections of the throat such as tuberculosis, syphilis, and growths are rare and almost always secondary. They give no difficulty in diagnosis, and the treatment must be directed to the primary conditions. Various neuroses of the throat may appear in the aged, most of them being secondary to cerebral or nervous disorders. Their treatment involves the treatment of the underlying condition.

Syphilis may manifest itself in the throat in the form of gummata which break down and ulcerate. The diagnosis is readily made by the history and by the presence of other tertiary lesions, while the Wassermann reaction is a conclusive test. The usual antisyphilitic treatment is indicated. For local treatment the ulcerated surface should be touched with a solution of nitrate of silver or protargol or argyrol. Primary and secondary lesions are rare.

Tuberculosis of the throat appears as a single or multiple ulcerations of the palate, pharynx or tonsils. They spread slowly by infiltrating adjoining tissue and do not heal readily. The tuberculin test may be necessary to determine their nature. Local treatment by the application of silver or zinc salts, and the constitutional treatment for tuberculosis is indicated.

Growths of the throat are very rare in later life and are almost always secondary. Little need be said of them as they are purely surgical conditions and their diagnosis is simple.

Neuroses of sensation and motion may occur, generally as part of psychic and nervous disorders, occasionally due to local irritation, as from tobacco, alcohol, hot food, ice, etc. Warm emulcent liquids may be employed to relieve hyperesthesia and

spasm, and if these fail, spraying with a 2 per cent. cocaine solution will generally give temporary relief. The cure depends upon the underlying condition.

LARYNGEAL DISEASES

Acute laryngitis may arise in the aged from the same causes that produce the disease in younger individuals. The most frequent cause, however, is spasmodic cough. The senile atrophic mucous membrane of the larynx is not readily stimulated to acute inflammatory activity and for that reason acute laryngitis is not as frequent as it is in younger individuals. The disease is much milder, there is rarely any elevation of temperature, the pain is not severe, and the feeling of some substance irritating the larynx, and inducing a cough, is not as pronounced as in the young. Hoarseness is, however, an early and persistent symptom and may proceed to complete aphonia. The laryngoscopic appearance of acute laryngitis presents redness and swelling but not as pronounced as in maturity; there is little mucus, and little change in the vocal cords. Unless soon relieved the disease becomes chronic, or by extension into the trachea and bronchial tubes, gives rise to a chronic bronchitis. The treatment is mainly hygienic, unless distressing symptoms appear. Rest in bed, warmth, a clear dry atmosphere, and abstaining from the use of the voice will generally effect a cure. Mild diaphoretics may be used and inhalation of a weak saline solution is often beneficial. If there is a persistent cough with scanty secretion, heroin and the syrup of senega may be used. Hot and cold applications and the applications of salt pork, etc., to the neck are concessions to the therapy of past ages. It is doubtful if these have any effect upon the disease.

Acute submucous laryngitis is an extension from the mucous inflammation. It is very rare and occurs only when a grave acute laryngitis has been produced by a powerful irritant and the irritation persists. It may lead to stenosis of the larynx and necessitate tracheotomy.

Perichondritis occasionally occurs in the aged, generally as a septic condition following infectious diseases, local ulcerations, metastatic abscesses, etc. It occurs in two forms, perichondritis interna and externa, the former consisting of an inflammation of the inner coat with swelling of the mucous membrane, the

latter as an inflammation of the outer coat with abscess formation. In perichondritis interna the symptoms are those of acute laryngitis, with progressive stenosis, dyspnea and hoarseness leading to aphonia. In perichondritis externa the early symptoms are pain and fever; later, an abscess on the larynx forms, which may break down and cause constitutional septic symptoms. As the disease is almost always of septic origin, serum therapy may be of service. If this fails surgical intervention becomes necessary.

Edema of the larynx may occur in the aged through impaired circulation in cardiac disease. It also occurs in nephritis, infectious diseases, chronic laryngitis, or as a result of the inhalation of irritating vapors, and other traumatic causes. It does not differ from the same disease in earlier life and must be treated the same way. When diaphoretics, diuretics and hydragogue cathartics fail, intubation or tracheotomy must be resorted to.

Syphilis of the larynx is rare and the cases that do occur are almost without exception tertiary gummata. If seen early and while the growths are still small, they will disappear readily under salvarsan followed by the mercury and iodine treatment. After they begin to break down into syphilitic ulcers, cure is somewhat more difficult. Under antisyphilitic treatment they will gradually diminish in size, however, and disappear, leaving scar tissue behind, which, upon contracting, produces stenosis. In rare cases the ulcerations of the cartilages will cause destruction of them and collapse of the larynx with asphyxia.

Tuberculosis of the larynx is very rare in the aged. It manifests itself in ulcers which may appear in any part of the larynx. It is often impossible to differentiate between a tubercular and a syphilitic ulcer. The latter is usually clean looking and not painful, while the tubercular ulcer is usually covered with caseous debris and is painful; the syphilitic ulcer has an excavated base with smooth everted edges, while in the tubercular one the edges are sloping and ragged. These distinctions, however, are not always well marked and the differential diagnosis will then depend upon the history, associated symptoms and signs, the result of antisyphilitic treatment, and finally, serum tests. The treatment consists of cauterization by silver nitrate, or similar silver salts, lactic acid or weak chromic acid solution, and the application of orthoform in 10 per cent. solution by

means of a spray. The systemic treatment of the underlying condition is necessary.

Neuroses of the larynx are infrequent and are then almost always associated with general psychic or nervous disturbances. *Anesthesia* is not recognized unless a laryngeal probe or other foreign body is introduced, when there will be found an absence of pain and reflex action. It is extremely rare, however. Galvanism is the appropriate remedy.

Hyperesthesia sometimes occurs in acute or chronic laryngitis. A slight irritation, such as a change in the temperature of the air, or a dusty atmosphere, will cause coughing, while any more severe irritation will cause laryngeal spasm.

The irritability can usually be allayed by spraying the larynx with a 10 per cent. solution of orthoform or a 2 per cent. solution of cocaine. *Spasm* of the larynx may occur from intense irritation, as from noxious vapors, dust, sudden temperature change in the inspired air, irritation of the vagus or of one of its laryngeal branches, excessive use of the vocal cords, or in hysteria or tabes. The treatment depends upon the cause. A whiff of chloroform may be required to allay a spasm. *Laryngeal paralysis* occurs occasionally in the aged. It may be due to hysteria or neurasthenia, bulbar paralysis, various spinal lesions, compression of the vagus or one of its laryngeal branches by growths, aneurysm, glandular enlargement, pericarditis, infectious diseases, poisons, muscle or nerve degeneration, cold, etc.

The symptoms depend upon the nerve and muscles involved and are mainly connected with phonation. These symptoms are almost all due to unilateral or bilateral paralysis of the abductors, adductors or tensors of the vocal cords. In paralysis of those muscles that are supplied by the recurrent nerve, the patient is voiceless and unable to cough. In paralysis of the abductors there is dyspnea. Unilateral paralysis of these muscles is extremely rare. The adductors and tensors are usually paralyzed together. If bilateral there is aphonia, if unilateral the voice is low and rough.

The treatment depends upon the causative condition. For local treatment galvanism, faradization, and vibration are of service. Inhalation of creosote, menthol and ammonia can be tried. Local applications are of doubtful utility.

DISEASES OF THE THYROID GLAND

Primary diseases of the thyroid are rare in senescence although some authors regard the normal senile degeneration of the thyroid gland as a form of myxedema. Horsley indicated many points of similarity between myxedema and the senile cachexia and thereon based his conclusion that the senile cachexia depends upon the degeneration of the thyroid gland—the more slowly this gland degenerates the slower the process of involution which causes the senile cachexia.

Myxedema is, however, a pathological condition in which the symptoms have but a superficial likeness to the senile cachexia and it is doubtful whether the myxedemic degeneration of the gland is identical with the senile degeneration. The characteristic symptoms of myxedema are swelling and infiltration of the subcutaneous tissue, dry scaly skin, general increase of the soft parts, and mental impairment. The skin of the face becomes swollen, especially about the eyes and chin, the nose and mouth become thickened and the face has a dull, heavy, expressionless appearance. The tongue becomes thick, and is protruded with difficulty. The hands and feet increase in size and may lose their contour. There is diminished surface sensibility, all other senses become blunted, the mind weakens, the will is impaired and responses to stimuli are slowed. These symptoms are sufficiently pronounced to distinguish it from senile cachexia. The treatment consists of the administration of thyroid gland or an extract of it which must be continued for weeks after the symptoms have disappeared or until palpitation of the heart announces that it has exceeded the limit of its therapeutic effect. To prevent a relapse a dose should be taken at regular intervals. Thyroid extract has no effect upon the senile cachexia.

Bronchocele and **exophthalmic goiter** are very rare in the aged, and they do not differ from the disease of earlier life. A bronchocele carried over from maturity may decrease in size and disappear in the process of involution without treatment.

Cancer of the thyroid may occur as a primary disease, giving the usual symptoms—namely, rapid increase in size, cachexia and infiltration of neighboring lymphatics. Kocher says it occurs most frequently in localities where goiter is endemic and attacks almost exclusively those in whom the thyroid is degenerated. The treatment is surgical.

Tuberculosis may occur as part of a miliary tuberculosis but caseous degeneration of the former as well as gummous degeneration of *syphilis* are very rare. *Acute thyroiditis* is rare and does not differ from the disease in early life.

DISEASES OF THE ADRENALS

Little is known of the diseases of these glands in the aged. *Addison's disease* has been noted, but it does not differ from the disease in earlier life. Grawitz has described a growth upon the glands, which is occasionally found in old people and which begins as a benign tumor but may become malignant. Cancer and other growths, such as caseous and gummous degenerations, have been observed as secondary conditions, but they give no distinctive symptoms apart from the symptoms of the primary disease.

ACUTE BRONCHITIS

Etiology.—Acute bronchitis in old age has a similar symptomatology to the same disease of earlier life. Owing to the atrophy of the mucous membrane in the aged, a much more powerful irritation is required to cause an acute inflammation and there is a greater tendency to involve the finer tubes and to run a chronic course. Great stress has been laid upon the influence of bacteria in the production of acute bronchitis and other acute inflammatory diseases. The senile organism is, however, more or less immune to bacterial influences, and when such infection does occur, it is either because the resistance had been lowered through disease or debility, or because the germs were exceptionally virulent. In either case the disease is much graver than in earlier life. When the inflammation is due to local irritation, and this is generally the case, it either subsides upon removal of the irritation, or it becomes chronic. Acute bronchitis also occurs frequently as a secondary infection in the course of an infectious disease and in these cases the infection rapidly involves the finer tubes and produces a bronchopneumonia.

Symptoms.—The symptoms of simple non-infectious acute bronchitis are much milder than in younger individuals. There is little or no pain nor any sensation of oppression in the chest, no fever, and little irritation, hence less tendency to cough.

The expectoration is scanty, thick, not purulent and is frequently swallowed. The physical signs are less marked. There are usually dry râles, but occasionally moist râles and prolonged expiration are found. If the capillaries are involved, these symptoms become aggravated, and there is a sense of oppression; dyspnea sets in, the cough is more severe, and powerful efforts must be made to loosen the tenacious mucus in the capillaries and to expel it. The respiratory murmur varies, it being weak or lost over a section in which the bronchial tubes are filled with mucus, and distinct where the tubes are clear. Fine and coarse, dry and moist râles are heard, fine moist râles being evident during inspiration over an area in which the capillaries are filled with mucus. Extension into the lung tissue may occur, producing bronchopneumonia. The infectious form of acute bronchitis begins with fever, headache and malaise. The local symptoms—cough, pain and expectoration—are more marked than in earlier life; the disease invades the bronchioles and finer capillaries and produces the infectious form of bronchopneumonia. An increase in temperature in a non-infectious case points to infection.

Treatment.—Simple non-infectious acute bronchitis requires no treatment apart from hygienic measures and the removal of the cause. Counter-irritants like mustard or surface hyperemia produced by dry cups over the chest, or hot foot baths, will hasten recovery. Of the expectorants the syrup of hypophosphite of ammonia liquefies the mucus, ipecac increases its flow, and senega acts as an irritant to the mucous membrane, thereby increasing the tendency to cough. The narcotics—morphine, codein, heroin and dionin—relieve the pain, but dull the sensibility and lessen the irritation, thereby preventing cough, which is necessary to remove the accumulated mucus.

While hygienic measures are of primary importance in simple acute bronchitis, drug treatment is more important in the capillary form. The choice of drugs depends upon the condition of the mucus and upon the ability of the patient to expectorate it. The ammonia salts, the carbonate, chloride and hypophosphite, liquefy the mucus; senega and apomorphine aid in its expectoration. Apomorphine cannot be used if the heart is weak. Ipecac, squills, and grindelia, all increase the amount of mucus and should be used whenever it is scanty.

Narcotics, if required, should be given in combination with the other drugs. In the infectious form the treatment of capillary bronchitis should be followed. Frequent percussion of the chest is necessary to recognize the presence of pneumonia. Inhalation of creasote, guaiacol or thiocoll is beneficial in this condition. The heart should be watched and strychnine given if it becomes weak. Hygienic regulations are rest, a dry equable atmosphere (free from dust and smoke) in a low elevation, warm baths, light foods, no alcoholics and no excitement.

Fibrinous bronchitis is extremely rare in the aged. When it does occur it does not differ from the same disease of earlier life.

BRONCHIAL STENOSIS

Etiology.—The caliber of the trachea or of a bronchial tube may be diminished in the aged by various conditions such as pressure from without, a hyperplasia of the lining membrane, scar tissue, a growth within, a foreign body or muscular spasm. Owing to the multiplicity of causes which can produce stenosis, the disease is not rare. Stenosis of the trachea occurs most frequently where goiter is endemic. Aspiration of foreign bodies such as inspissated mucus or food particles is a frequent cause of bronchial stenosis in the aged. Pressure from without may be caused by a growth, aneurysm, enlarged gland, or by traumatism. Inflammatory swelling of the lining membrane is rare and scar tissue and growths in the trachea or bronchus are likewise very exceptional. Spasm may occur in bronchial asthma, in hay fever and as a result of intense irritation.

Symptoms.—Difficulty in respiration is the most prominent symptom. This may occur slowly, rapidly or suddenly, depending upon the cause. If mild, it gives trouble only upon exertion; if severe it produces a marked dyspnea, and if complete it causes asphyxiation, depending upon the location and extent of the occlusion. It is often difficult to determine the cause of dyspnea or the exact place of the stenosis. Sudden dyspnea occurs in spasm and in occlusion caused by a foreign body. Dyspnea without cough occurs in compression stenosis. In stenosis of the trachea or bronchial tube, however, the cause is usually evident and the location can be determined thereby. Upon auscultation there is normal respiration below the point of

contraction, a whistling sound at the point, and weak, higher pitched respiratory sounds above. In stenosis of a fine tube, atelectasis of the part of the lung supplied by the occluded tube may occur.

Treatment.—Treatment depends upon the cause. Surgical measures are generally required.

PERICARDITIS

Acute pericarditis is rare but pericardial adhesions are not infrequent. In most cases these adhesions date from early age when the pericarditis appeared as a complication of or following an acute articular rheumatism or some other infectious disease. In some cases there is a history of cardiac disease, in others a nephritis was the immediate precursor of the pericarditis. The acute disease in the aged does not differ from the one of earlier life. It begins as an adhesive pericarditis followed by a serofibrinous exudate. There is dulness on percussion, the apex impulse is weakened or absent, and fever, pain and dyspnea are present. A friction sound over the heart, synchronous with the heart contractions and not influenced by respiration, is pathognomonic of pericarditis, but this friction sound may be absent in the presence of an extensive exudate. The disease is more serious in the aged than in younger individuals, as it is usually associated with acute endocarditis and myocarditis arising from the same etiological factors that occasion the pericarditis. Recovery from the acute disease is rare and is then always followed by a chronic adhesive pericarditis with obliteration of the pericardial sac or adhesion to the pleura or chest wall. When the layers of the pericardium are adherent to each other there may be no symptoms at all or only friction sounds. When the pericardium is adherent to the pleura or chest wall there is displacement of the heart and consequent disturbance of its action. Prominent symptoms are a dimpling or retraction over the apex beat at each systole, the paradoxical pulse and a diffuse diastolic impulse. The symptoms are more pronounced in adhesion to the vertebræ. There is then a considerable hypertrophy, which is generally followed by dilatation of the heart with its complications.

The treatment of acute pericarditis in the aged comprises

rest and attention to the symptoms. The iodides may be given internally and a hyperemia may be produced over the region of the heart if there is much exudation, and if this fails it may be necessary to withdraw the exudate through a pericardial canula, always a dangerous operation in the aged. For a pericardium with adhesions to the chest wall, Brauer suggested a resection of portions of the ribs with separation of the adhesions. Drugs are useless in these cases except to relieve symptoms and to temporarily stimulate the heart.

Chronic mediastinitis usually accompanies a chronic pericarditis but it gives no distinctive symptoms and it is treated as part of the pericarditis.

GASTRIC ULCER

Gastric ulcer is rare after the sixtieth year. In its etiology and pathology it does not differ from the same disease of earlier life but the symptoms in advanced age may be modified by the changes in the stomach walls.

Symptoms.—Gastric ulcers have been found upon autopsy which gave no symptoms during life, while in other cases the first indication of an existing ulcer was a fatal hemorrhage or gastric perforation. In maturity the classical symptoms are pain, hematemesis and hyperchlorhydria. In the aged, normal or subnormal acidity occurs more frequently than hyperacidity, the pain is often slight and may not occur until two or three hours or more after taking food. There is a persistent ache, however, which only gives way to the more acute pain that follows the ingestion of food. This pain is rarely paroxysmal, but becomes gradually worse, until it has reached its maximum intensity, then it lessens, leaving an ache which persists. The pain is usually localized over the site of the lesion, most frequently in the median line below the sternum.

The hematemesis is rarely severe, yet it is a grave symptom. The aged do not vomit readily and when it occurs it signifies a severe irritation or hemorrhage. A severe gastric hemorrhage may be rapidly fatal or cause a cachexia from which the patient does not recover. Generally, there is a slight regurgitation of food an hour or two after eating, and in the matter brought up there will be a trace of blood, either as a small black clot or as a

particle of food streaked with blood. In suspected ulcer the food thus brought up should be carefully examined for this sign of the disease. Blood can sometimes be found in the feces, but it is then impossible to determine its exact source. The appetite is not impaired and there may even be a bulimia. The ingestion of food generally gives temporary relief from pain but the knowledge that the pain will appear later produces a fear of food. In many cases there is pyrosis, flatulence, eructations of gas, constipation, etc.

The only diseases which may give similar symptoms are cancer of the stomach, and ulcer of the duodenum. The pain of cancer is not as sharp, but it is more persistent, and occurs soon after the ingestion of food, the hemorrhage is darker, "coffee ground vomit," and there is a pronounced hypochlorhydria with the presence of lactic acid. There is progressive cachexia, glandular involvement and later a tumor can be felt at the site of the cancer. In duodenal ulcer the pain occurs several hours after eating, and it radiates to the back on both sides of the spine. Food and alkalies give relief from pain while flatulence increases it. The tender point is usually about the umbilicus. Vomiting is rare and does not give relief from pain. Jaundice is occasionally present and blood is often found in the stools.

Other diseases giving gastric pain, like gastralgia, the gas ric crises of tabes, acute gastritis, etc. have pathognomonic symptoms, or histories, or are not accompanied by hematemesis, while cirrhosis of the liver, in which there may be vomiting of blood, has no gastric pain. Erosion of the mucous membrane of the stomach, which has been considered a preliminary stage of gastric ulcer, has not been observed in the aged (*Ewald*).

Treatment.—The treatment of gastric ulcer does not differ from the treatment of this disease in younger persons. The most important indication is to prevent further irritation of the lesion and this can be done only by withholding all food as long as possible and resorting to rectal feeding. This can generally be done for three or four days, when bland articles of diet, such as calf's-foot jelly, oat-meal gruel, milk, and malted milk may be given. In the meantime bismuth subnitrate in 10-grain doses combined with an equal quantity of magnesium carbonate should be given three or four times a day. The treatment

should be continued for a week and afterward more substantial food may be permitted. Alcoholics, spices, and acids must be avoided and as little salt as possible should be taken. If the pain is severe hypodermics of morphine and atropia may be used. Cocaine will allay the irritability which leads to vomiting, but unless there is nausea, it should not be used. Severe gastric hemorrhage and perforation are almost invariably fatal. If hemorrhage occurs, tannic or gallic acid may be used as astringents and pieces of ice should be swallowed. The danger in these cases is more from shock than from the amount of blood lost and the shock should receive attention as soon as the patient has received the ice or the astringent. The most rapid and effectual treatment is a hypodermic injection of 30 minims of ether. Surgical intervention may become necessary.

DUODENAL ULCER

Duodenal ulcer is rare after the sixtieth year. Its etiology and pathology is the same as in younger individuals.

Symptoms.—Symptoms are generally vague and the diagnosis must often be determined by excluding gastric ulcer, gastric cancer, gall-stone colic, intestinal colic and peritonitis. The pain is localized about the umbilicus, and appears from three to four hours after eating. It is relieved by food and by alkalies. There may be griping pains, after the pains produced by the chyme and the discharge of acid into the duodenum cease. Pressure over the site of the ulcer intensifies the pain. Vomiting is rare, but the feces generally contain a trace of blood. Flatulence produces a sharp pain. Jaundice is sometimes present and the stools are then clay-colored.

Treatment.—The treatment is as for gastric ulcer, including surgical intervention, but predigested foods can be given throughout the disease and alkaline mineral waters are admissible.

ENTERITIS

Under this name will be described acute and chronic inflammations of the intestines including localized inflammations such as colitis, typhlitis, proctitis, etc. It is often impossible to localize an intestinal inflammation and in many cases of

enteritis more than one portion of the bowel is involved. Inflammation of the rectal wall can usually be diagnosed by inspection; inflammation of other portions of the bowels rarely give such clearly defined symptoms that their exact location could be determined. They will, therefore, all be included under one head, and where localized inflammations present special symptoms these will be mentioned.

ACUTE ENTERITIS

Etiology.—According to Ewald an acute interitis can occur only in the healthy senile individual. If there is the ordinary senile degeneration of the intestines present, the acute inflammation becomes converted into a chronic one as soon as the acute symptoms lessen in severity.

The most prolific cause of acute enteritis in the aged is improper or excessive food. Owing to the lessened peristaltic activity, food remains longer in the bowel and, owing to the diminished bile and intestinal secretions, it decomposes more readily and this decomposing material irritates and inflames the lining membrane of the intestines. The most common food articles that rapidly decompose are cold storage meat and eggs and canned foods, over-ripe fruit, and tainted milk. Certain articles of food will produce in some persons a catarrhal condition with diarrhea, and the change from hard to soft drinking water will often give the same effect. The prolonged use of drugs, especially of inorganic salts, will cause an acute enteritis, although generally by the time this enteritis is recognized it has entered a chronic stage. A change or deficiency in the gastric or intestinal secretions which permits undigested food to pass into the lower bowel will cause an enteritis which usually begins and progresses so mildly that it becomes chronic before it attracts attention. Only a sudden and profound change in the secretions will produce an acute inflammation with acute symptoms. Infection, the invasion of animal parasites, chilling of the surface of the body and nervous influences, such as shock, fear or other strong emotion, may cause an acute catarrhal inflammation of the intestines. An acute enteritis may be secondary (1) to an inflammation or ulceration, (2) to gangrene or cancer in an adjoining tissue which by extension has involved

the bowel, (3) to an acute infectious disease, or (4) to local circulatory disturbances.

Pathology.—Owing to the physiological senile degeneration of the mucous membrane of the intestinal tract, the inflammatory changes as found on abdominal section in maturity are mild or absent in senility. There may be a slight hyperemia and an increased flow of mucus from enlarged mouths of mucous glands, more often there are areas showing desquamation of epithelial cells, enlarged follicles, and areas of ulceration.

Symptoms.—The most prominent symptoms of acute enteritis are pain and diarrhea. The pain is usually colicky, coming on spasmodically and is partly relieved by pressure. An inflammatory pain, continuous and intensified by pressure, indicates an extension of the inflammation to the peritoneum or other viscus. If the upper bowel is affected the pain is more severe and persistent. In inflammation of the jejunum or ileum the pain is most severe at the umbilicus. In the lower bowel it is rather a dull ache, rarely intense or paroxysmal, but tenesmus is usually present. In proctitis there is always tenesmus, a burning sensation in the rectum and, if the inflammation is due to a foreign body or to a hard mass of feces pressing against the sphincter, there is a sharp, cutting pain. In typhlitis the symptoms are referable to the appendix which is usually involved also (see appendicitis). In cases where improper or excessive food is the cause, the pain comes on suddenly two or three hours after the food has been taken. When due to a toxin it may come on in a few minutes after ingestion of food. If due to cold the pain is usually mild and if due to nervous influence there is frequently entire absence of pain. Tenesmus is always due to rectal irritation.

Diarrhea is the most distinctive symptom of enteritis, yet it may be absent. The stools are at first soft, then watery, the normal intestinal contents being carried away by the first few movements. Much can be learned from the character of these evacuations. If the early ones contain feces in small lumps, the upper bowel is involved; if the feces are formed then the trouble lies in the descending colon or rectum. Mucus generally indicates inflammation of the large intestines though minute particles may come from the small bowel. Formed feces covered with mucus indicate proctitis. Cecal mucus is

generally dark and jelly-like, while the color becomes lighter and the consistency more fluid the nearer the inflammation is to the anus. Strips or bowel casts, indicating a mucous colitis, are rare. Blood is seldom found in the enteritis of the aged. When it is present it points to a serious complication, or to dysentery. A sour-smelling stool is due to excessive carbohydrate fermentation; a foul-smelling one indicates intestinal decomposition or else dysentery or carcinoma. Pus is found in ulcerative enteritis. Food remnants appear in the stool if the duodenum is involved. Yellowish, greenish, or grayish dejections come from the upper bowel. In the enteritis due to cold or nervous influences, the dejections are watery and generally odorless, passing without pain or tenesmus. In almost all diarrheas the discharges irritate the rectum and will sooner or later produce tenesmus.

In addition to the pain and diarrhea there are generally tympanites, borborygmi, sometimes cramps in the muscles of the abdomen and legs, vomiting and intense thirst but no elevation of temperature unless there is some infection. Vomiting is infrequent in the aged. The urine becomes scanty and high colored and may contain a trace of albumin and casts. If the small intestines are affected there will be an increase of indican. The symptoms are usually more severe than in earlier life, although the alvine discharges may be reduced in quantity and frequency owing to the atrophy of the lining mucous membrane and glands. In many cases of acute enteritis there is rapid exhaustion which may terminate in collapse and death.

Treatment.—The principal indications for treatment are the diarrhea and pain. The measures for the relief of the diarrhea depend in part upon the location and in part upon the cause of the enteritis. In all cases it is necessary to secure thorough evacuation of the bowel before checking the diarrhea. If the inflammation is in the rectum this can best be accomplished by a high enema using an alkaline solution. If in the upper bowel, castor oil should be used, but if this cannot be taken another vegetable cathartic, preferably rhubarb or cascara, should be used. These drugs act slowly, evacuation following in from twelve to twenty hours. If rapid action is required we must employ the saline cathartics in large doses and given in hot water. In many cases the removal of offending

material from the bowels and rest will relieve the diarrhea and if care is taken with the diet for a day or two there will be complete recovery from the enteritis. If the diarrhea is due to cold, the application of moist heat will give relief, and if due to sudden emotion the relief of the nervous symptoms is generally all that is necessary to check it. When diarrheal discharges continue after the bowels have been cleared, they should be checked by the use of astringents, preferably bismuth subnitrate in 10-grain doses. Opium in 1/4-grain doses should be added if there is much pain. For the pain alone a hypodermic injection of morphine and atropine can be used. For the relief of tenesmus a suppository of extract of belladonna and opium gives relief. The powerful mineral astringents, like sulphate of copper or zinc, are rarely required, but if the discharges continue to have a foul odor the sulphocarbolate of soda or zinc should be used. All astringent drugs should be discontinued as soon as the diarrheal discharges cease. Incidental symptoms, such as tympanites, borborygmi and muscle cramps pass away as soon as the bowels have been evacuated. Thirst should be relieved by ice, not by excessive draughts of water. The danger from exhaustion and collapse is much greater than in younger individuals. In these cases brandy acts well and strychnine should be given if the pulse becomes weak.

During an attack of acute enteritis the food should be light, bland, fluid and preferably predigested. Food liable to decompose or ferment in the intestines should be prohibited and on the first day of an acute attack all food should be avoided. After the first day small quantities of some predigested food may be given for a day, after which more substantial nourishment can be permitted. Cold storage meat and canned food should never be used in cases showing a tendency to diarrhea or flatulence. If the diarrhea is followed by constipation, mild vegetable laxatives should be employed.

CHRONIC ENTERITIS

Etiology.—Chronic enteritis in the aged is usually secondary to an acute attack. A slow, progressive enteritis is produced when undigested food passes into the lower bowel or when there is excessive intestinal fermentation and decomposition. The

prolonged use of inorganic salts may produce a slow progressive enteritis. It may also occur as a secondary condition following intestinal ulceration or other lesions, or may be due to chronic circulatory disturbances or to diseases of metabolism.

Pathology.—In mild cases no changes can be found. There is usually some thickening of the mucous membrane with erosion and pigment deposits in or around the follicles. Passive hyperemia with ecchymotic spots is sometimes found, and occasionally there are bands of dark thickened, or of light atrophied, membranes between them.

Symptoms.—The most prominent symptom of chronic enteritis is a diarrhea alternating with constipation. In the progressive form there is a gradual increase in the number and fluidity of the stools, these occurring most frequently in the morning. The character of the stools is the same as in acute enteritis and there are often the same incidental symptoms, flatulence, tympanites, borborygmi, and pain. The pain, however, is usually slight, rarely colicky. If ulcerations are present there will be tenderness over the site of the ulcer and pain on pressure. Localization of the inflammation is difficult, yet the determination of its place is necessary to secure a proper dietary. Lientery points to duodenitis and this diagnosis is confirmed if there is jaundice and pain at or above the umbilicus.

In inflammation of the ileum or jejunum there is also some pain or tenderness about the umbilicus with occasional colicky pains, the stools are fluid, grayish or greenish, and have a sour or foul odor. The pain comes on two or three hours after eating. A typhlitis gives a brownish partly formed thick stool covered with a dark jelly-like mucus, pain is in the right groin and the feces have the usual fecal odor. Symptoms of appendicitis may be present. Colitis produces a large fairly formed stool covered with a light mucus and the pain comes on a few minutes before the stool is passed. The pain can be more readily localized in the ascending, transverse or descending portion of the colon than in any other part of the bowel.

Proctitis has an almost pathognomonic symptom, tenesmus, with painful dejections. The stools are formed and are covered with mucus. The presence of pus, blood, or shreds of mucus in the dejections indicates an ulceration, and the location can generally be determined by the intense pain produced when

pressing upon it. It is important to differentiate between syphilitic, tubercular and simple ulcerative enteritis, but the former two can generally be diagnosed by the history and attending symptoms. Carcinoma of the intestines may simulate chronic enteritis, but the intense pain, presence of tumor, cachexia and involvement of other tissues will serve to differentiate it from the milder affection.

Treatment.—The treatment is primarily dietetic. The diarrhea can in most cases be temporarily controlled by intestinal astringents, or, if the fault lies in the rectum or colon, by starch enemata. The most important point in the treatment of chronic enteritis is to avoid the introduction of irritating substances into the bowel. Food liable to ferment or decompose, strongly acid substances, and food containing much indigestible matter must be avoided. An examination of the stools should be made, and if food particles are found, such substances must be avoided or given in a predigested form. Especially objectionable on account of their indigestibility are vegetables containing much cellulose, meat containing much cartilage, tendon or connective tissue, skin and fatty smoked meats. Readily decomposing foods are cold storage meat and eggs, all canned food, and overripe fruit.

Medicinal measures are confined to intestinal astringents and antiseptics, and for the occasional constipation, mild vegetable laxatives. Proctitis alone can be treated locally by astringent lotions and enemata and, if there is much pain or tenesmus, by cocaine and belladonna suppositories.

DISEASES OF THE LIVER

Cirrhosis of the liver is infrequent after the sixtieth year and the hypertrophic form is extremely rare in advanced life. The infectious diseases play an insignificant rôle in the etiology of cirrhosis of the aged, and owing to the natural atrophy of the organ, the enlargement in the hypertrophic stage is not marked. For a similar reason the enlargement of the senile spleen, which is a prominent symptom in earlier life, rarely reaches the size of the normal organ in maturity. Gastrointestinal disturbances arise early and there are, occasionally, gastric and intestinal hemorrhages and frequently bleeding hemorrhoids. Ascites,

which occurs late in maturity, occurs early in senility, but rarely reaches the extent seen in earlier life. The general disturbance of the circulation and nutrition causes a more profound senile cachexia, the complexion being sallow or tending toward jaundice with cyanotic patches about the nose and cheeks. There are occasionally shooting pains, and more frequently tenderness upon pressure over the liver. Cerebral symptoms develop late in the disease.

A positive early diagnosis of hepatic cirrhosis in the aged is impossible, the early symptoms being usually referred to the stomach and bowels. The history of the prolonged use of alcohol on an empty stomach is suggestive, and the enlargement of the liver points to cirrhosis. A definite diagnosis can be made later when atrophy sets in and ascites and gastric or intestinal hemorrhages occur. Primary cancer of the liver in an alcoholic subject may be mistaken for cirrhosis in the hypertrophic stage. If nodules cannot be felt it may be impossible to differentiate them until secondary symptoms of the cancer, or atrophy and ascites of cirrhosis, appear. Cancer of the liver progresses more rapidly than cirrhosis, the cachexia is more marked and appears earlier, and there is no ascites. Cardiac disease and peritonitis often present some of the symptoms of cirrhosis, but they have some pathognomonic symptoms of their own. The treatment of cirrhosis of the liver is unsatisfactory. Drugs have no effect, but may relieve the associated symptoms. A salt-free diet will sometimes retard the anasarca but where it fails, diuretics, hydragogue cathartics and diaphoretics must be used. The question of early or late paracentesis depends upon attending circumstances. Some authors suggest tapping at the earliest possible moment to prevent extreme distention and weakening of the abdominal muscles, others suggest that it be deferred until the dyspnea, cyanosis or pulmonary edema endanger life. Diuretics are usually ineffectual in extreme ascites and hydragogue cathartics may cause exhaustion. It seems best to tap early and reduce the edema by diuretics, repeating the operation whenever the abdomen is again distended, and not to delay until dyspnea or cyanosis make it imperative. A milk and simple vegetable diet, the administration of simple bitters with the addition of hydrochloric acid, and but little liquid food with the exception of milk are the principal dietetic regulations. Alcohol

must be absolutely prohibited. Other treatment is purely symptomatic.

Hypertrophic cirrhosis, *Hanot's cirrhosis*, is extremely rare. It resembles the hypertrophic stage of atrophic cirrhosis, but the liver does not atrophy, there is no ascites, though there is jaundice but without the clay-colored stools that occur in other diseases that are accompanied by hepatogenous jaundice. The disease is slowly progressive and incurable. The treatment is symptomatic.

Syphilis of the liver appears as a tertiary, diffuse, interstitial hepatitis, or as gummata. In the former case the disease resembles atrophic cirrhosis with but slight ascites, and cachexia, in the latter case there are growths upon the liver which can usually be felt, they may produce pressure symptoms, but cachexia is slight. In both cases there are symptoms of tertiary syphilis in other tissues. The history and the Wassermann test will determine the diagnosis. The usual antisyphilitic treatment is indicated.

HYPEREMIA OF THE LIVER

Active hyperemia may be physiological, as when occurring after a full meal, or pathological when due to infectious and toxic diseases. The later is rare in the aged and when it occurs it lasts as long as the underlying disease lasts. There is a sense of oppression, tenderness on pressure, enlargement of the organ, sometimes jaundice and diarrhea. The treatment depends upon the cause. Small doses of calomel may relieve the congestion temporarily.

Passive hyperemia: Etiology.—This is a common ailment in the senile and generally occurs with cardiac dilatation or with other cardiac diseases. Anything which obstructs the flow through the vena cava or hepatic vein will cause hepatic venous stasis.

Pathology.—The liver passes through several stages beginning with enlargement due to venous engorgement, followed by hyperplasia of connective tissue and pigmentation of the cells, producing the nutmeg liver, and lastly degeneration of the cells and atrophy, forming the atrophic nutmeg liver. The disease

producing no discomforts until a later stage, the physician seldom sees the patient during the first stage.

Symptoms.—During the early stages there may be a sense of weight in the region of the liver, tenderness on pressure and enlargement of the organ, but the early symptoms are not well marked, the liver being normally contracted in advanced life. Later there is impairment of the functions of the organ, exhibited in gastrointestinal disturbances, clay-colored stools, constipation, dark urine, sallowness or jaundice and cachexia with mental depression. The liver is diminished in size and tender.

Treatment.—The treatment depends upon the underlying condition. Calomel, blue mass or bile salts are indicated internally, and counter-irritation externally, by sinapisms, leeches, dry cups, etc.

Abscess of the liver is rare. The *solitary* or *tropical abscess* is occasionally found in dwellers of warm countries and usually follows amebic dysentery. *Multiple* or *pyemic abscesses* occur in pyemia. Embolic abscess, a solitary abscess, is extremely rare. The symptoms are the constitutional symptoms of septic infection with local symptoms of pain, tenderness, sometimes a feeling of weight or dragging when lying on the left side, enlargement of the organ, occasional digestive disturbances, dyspnea, cough, ascites and jaundice. Diagnosis of multiple abscess is often difficult as the local symptoms may be mild and escape notice. The disease may be mistaken for empyema, but the latter disease has earlier symptoms of pleurisy.

Rupture gives symptoms of shock and profound sepsis.

Treatment is surgical. Serum therapy may be tried for the systemic disease.

Fatty degeneration may occur as part of general obesity, as a result of alcohol or poison, or in the course of some infections, and in cachexias. The disease gives no distinctive symptoms. The liver may be enlarged, but is not nodular or painful and there is no jaundice or cachexia unless these are due to a primary disease. The treatment depends upon the underlying condition.

Amyloid degeneration is very rare in the aged. The liver becomes greatly enlarged and the border can be felt as a sharp ridge but it gives no distinctive symptoms. The treatment depends upon the cause.

DISEASES OF THE PERITONEUM

Primary Acute Peritonitis.—It has been declared that a primary acute non-infectious peritonitis does not occur. Cases of acute peritonitis following traumatism, however, do occur without any evidence of bacterial action, though such cases are extremely rare.

Secondary acute peritonitis may be due to extension of an inflammation from an adjoining tissue, or to a perforation into the abdominal cavity. This disease, which is infrequent in the aged, does not differ from the peritonitis of earlier life except that the local symptoms are milder, and the constitutional symptoms are graver, while the disease is almost invariably fatal. The exudate is fibrinous, serofibrinous, hemorrhagic, purulent or gangrenous. In some cases there is no pain except upon motion, and cases are found without elevation of temperature. In severe cases there is complete anorexia and insomnia, which often cannot be relieved by drugs.

In senile cases the invasion is frequently gradual, without chills, and with but little pain, which, however, rapidly increases in severity, while several days may elapse before the abdomen becomes distended. In these cases the peritonitis is an extension of an inflammation from a neighboring inflamed tissue. Constitutional symptoms of septic infection are present in many cases. Peritonitis is differentiated from intestinal occlusion by the fever and pain which are present at the onset, while in obstruction the pain appears later and complete constipation is present from the first. There is besides generally a history leading to the production of the disease which will aid in the diagnosis. The disease is usually fatal in a few days. Serum therapy may possibly hold a cure for this as for other infectious diseases. Surgical measures have availed in some cases in earlier life and as a last resort may be tried in the aged. The usual treatment with opium relieves the distress but does not cure. It is hardly necessary to mention the numerous remedies that have been proposed for the treatment of peritonitis as none are curative. In rare cases where the peritonitis comes on slowly as a result of an inflammatory extension from an adjoining organ, absolute rest will be followed by a subsidence of the acute symptoms and the disease may become chronic.

Chronic peritonitis may follow an acute attack, or a prolonged peritoneal irritation, such as in ascites, or it may be due to tuberculosis or cancer. Occasionally, after abdominal section, the symptoms of a mild peritonitis appear, probably as a result of the irritation of the peritoneum during the operation.

The symptoms are the same as those of acute peritonitis but are less intense and more prolonged. The constitutional symptoms are mild, or absent or masked by the more pronounced constitutional symptoms of the underlying disease. The course of chronic peritonitis is a slowly progressive one unless due to cancer when the progress is usually rapid. The exudate may form bands or adhesions and cause constriction or displacement of organs and tissues, producing intestinal obstruction or occlusion, displacement of the ovaries, uterus, stomach, liver, or other abdominal organs. Many uninterpretable symptoms are found upon autopsy to be due to peritoneal adhesions with consequent displacement of abdominal organs.

The diagnosis is generally based upon the history. If there is any question between chronic peritonitis and ascites, it will be necessary to resort to paracentesis and consequent examination of the exudate. The serous exudate in peritonitis has a high specific gravity (1015–1024, Ewald) and it has a decided tendency to coagulation and production of fibrin. The serous exudate of ascites has usually a specific gravity below 1015 and may remain fluid. Cysts are rare in the aged and there is usually a localized swelling in a region in which peritonitis is infrequent. An ascites and a peritonitis may exist at the same time, the irritation produced by the ascites causing peritonitis.

There is no cure for this condition. The symptoms may be relieved and the fluid withdrawn, but its withdrawal increases the danger from peritoneal adhesions. If these adhesions cause intestinal occlusion, or other grave symptoms, operation may be necessary.

Ascites is a symptom of numerous pathological conditions which cause disturbance in the abdominal circulation, or produce a general hydremia. The most frequent of these in old age are diseases of the heart and lungs which cause passive congestion of the abdominal vessels, obstruction of the portal circulation, chronic peritonitis, nephritis, cancer, and the pressure of

tumors. Tubercular, chylous and adipose ascites are extremely rare in the aged.

The diagnosis of a collection of fluid in the abdominal cavity ought to give no difficulty. Cysts are rare in the aged, and are unilateral and localized in the organs which contain them. Bladder distention has been mistaken for ascites—an unpardonable error. The treatment of ascites depends upon the cause. The local treatment is by paracentesis. This should be done as early as possible and the fluid withdrawn slowly, care being taken to first empty the bladder. It is often possible to retard future exudation by the use of diuretics, diaphoretics, and hydragogue cathartics, the selection being determined by the condition of the heart, kidneys and general strength of the patient.

DISEASES OF THE PANCREAS

Apart from cancers which, according to Ewald, form about 60 per cent. of all pancreatic diseases, the most frequent pathological condition of the pancreas found in advanced life is the **chronic pancreatitis** associated with diabetes mellitus. In this disease there is a proliferation of the interstitial connective tissue and degeneration and atrophy of glandular tissue. There are no distinctive symptoms. Usually, however, we find gastric and intestinal indigestion, emaciation, sometimes jaundice, epigastric pain, lipuria, glycosuria, and fatty stools. In rare cases it is possible to feel the indurated pancreas, thereby confirming the probable diagnosis. There is no curative treatment for this condition. The relief of symptoms and the administration of diastase, bile salts and pancreatin with all starchy food is the most that can be done.

Acute Pancreatitis.—This may occur as a hemorrhagic or suppurative pancreatitis, both exceedingly fatal conditions. It begins with intense pain in the region of the pancreas, vomiting and collapse. The abdomen becomes distended and tender and there is usually constipation. The sudden onset of the disease without previous illness or history distinguishes it from intestinal perforation, intestinal obstruction, perforation of a peptic ulcer and from gall-stones. Death usually occurs in a few days.

DISEASES OF THE SPLEEN

Primary diseases of the spleen are extremely rare and secondary diseases are infrequent. The *acute swelling* of the spleen that generally accompanies inflammatory and infectious diseases is not as pronounced as in earlier life and can rarely be diagnosed as the organ is physiologically atrophied and even marked enlargement will not reach the normal size of maturity.

The same applies to the *chronic enlargement* that accompanies diseases of the liver and also to local passive hyperemia in circulatory disturbance. Such swellings of the spleen produce no symptoms. The extreme swelling of *splenomegaly* may cause pressure symptoms but this condition is very rare in the senile. *Acute splenitis* occurs usually as part of the systemic disturbances occasioned by acute infectious diseases; rarely as the result of trauma or of extension of an inflammation from an adjoining viscus. It is generally accompanied by a *perisplenitis* and is then painful upon pressure over the spleen. The treatment depends upon the underlying cause. Local applications of hot moist cloths will relieve the pain. *Chronic splenitis* may follow an acute splenitis, while a gradual induration with hypertrophy may occur in the course of a chronic infection or disease of the liver. In this, as in all diseases of the spleen in which this organ is enlarged, excepting splenomegaly, the hypertrophy of the atrophied gland rarely reaches in size the normal volume of the gland in maturity. Consequently there are absent the sense of weight and oppression and the pressure symptoms that are characteristic of the enlarged gland in earlier life. In inflammatory conditions the location of pain may clear up the diagnosis. *Infarction of the spleen* may occur in the aged but there are no distinctive symptoms. *Abscess* usually follows an infarct and may give symptoms of a mild septic infection, the only symptom indicating its location being tenderness upon pressure over the organ. The treatment is surgical. There is no record of the result of serum therapy in this condition although its employment would be in harmony with its use in other cases of septic infection.

Splenoptosis occurs frequently as part of the general visceroptosis of advanced life. It gives no marked symptoms and is generally discovered accidentally while percussing the abdomen.

The displacement is most pronounced in women who were accustomed to tight lacing. If the displacement produces much discomfort, it can generally be relieved by a binder with a pad which will hold the organ in place.

Other conditions, such as tuberculosis, syphilis, growths, amyloid degeneration, cysts, etc., are extremely rare in the aged. When present they are usually secondary and give no distinctive symptoms apart from the symptoms of the primary disease.

DISEASES OF THE KIDNEYS

Acute or **active hyperemia** of the kidneys is generally due to irritation from drugs, while toxemia from infectious diseases, the most frequent cause in earlier life, is infrequent in the aged and acute parenchymatous nephritis, in which acute hyperemia is the initial condition, is extremely rare. There are no clearly defined symptoms of acute hyperemia beyond a pain or a dull ache over the kidneys which is increased upon pressure. The urine generally contains blood and albumin but no casts. The diagnosis must be made from these symptoms and from the history of the ingestion or use of irritating drugs. The treatment demands the removal of the causative irritation, and the administration of large quantities of alkaline water.

Chronic or **passive hyperemia** occurs frequently in the aged as a result of impaired abdominal circulation caused by disease of the heart, lungs, liver, or pressure from growths. Arteriosclerosis may be a causative factor, although the usual result of this condition is an anemia with consequent atrophy, due to malnutrition. The kidney in passive hyperemia is enlarged, congested and deep red in color, darker in the pyramids than in the cortex. There is no marked increase in connective tissue and no degeneration of the epithelium of the tubes. The symptoms are almost exclusively associated with functional activity, as evidenced by the urine. The quantity is diminished, the specific gravity is increased, and may reach 1030 or more, the solid constituents are increased, the percentage of urea and uric acid being high. There is usually albumin but rarely blood or casts. The diagnosis is readily made from the examination of the urine. The treatment depends upon the cause. *No irritant diuretics* should be given.

Anuria or total *suppression of urine* may occur in the course of any renal or infectious disease, or as the results of traumatism, shock or occlusion of both ureters. It may arise suddenly as in shock, rapidly as in infectious disease or slowly, the amount of urine decreasing day by day until there is complete anuria, as in chronic hyperemia. Unless speedily relieved uremia follows but recoveries have been recorded even after total suppression lasting fifteen days. Treatment depends upon the cause. In some cases vegetable diuretics will produce such irritation that free diuresis is followed by complete suppression. In such cases bland or lithia water may be taken and at the same time hydragogue cathartics and diaphoretics should be given to remove the excess of fluid. In some cases hot baths or the Turkish bath will stimulate all excretory secretions. Generally vegetable diuretics like uva ursa, buchu, digitalis or juniper are required. If there is edema or symptoms of approaching uremia a salt-free diet should be gradually instituted and more powerful diuretics such as potassium acetate or nitrate, and hydragogue cathartics should be used.

Uremia is usually of the chronic type, is associated with arteriosclerosis or chronic interstitial nephritis, and is accompanied by a persistent watery diarrhea. The gradually increasing nervous and cerebral symptoms ending in convulsions and coma, and the diminished excretion of urine and solids determine the diagnosis. Mild symptoms may persist for years, with acute exacerbations, during which convulsions, dyspnea, Cheyne-Stokes respiration, and coma may occur. Cases are occasionally met with which give a number of vague symptoms, such as headache, insomnia, vertigo, neuralgic pains, increasing mental dulness, impairment of sight, hearing and other senses, various gastric and intestinal disturbances, etc., and a diagnosis of general arteriosclerosis is made until a uremic coma suddenly discloses the underlying condition. In all such cases it is necessary to determine the amount and character of the urine for several days in succession in order to make a correct diagnosis, yet this is rarely done. The most important part of the treatment of chronic uremia is a salt-free diet which in the aged, must be gradually introduced. Diuretics should be used to increase the action of the kidneys and diaphoretics and saline cathartics should be given to increase elimination of

waste. If there is a nephritis present the saline diuretics should be used instead of the irritating vegetable ones. The oils and balsams are contraindicated. Pilocarpine is dangerous if the heart is weak. Narcotics may be required for convulsions.

Albuminuria in the aged if not in large amount and not associated with casts is generally of no importance and indicates only a senile, contracted kidney. It is, however, necessary to exclude other causes for albuminuria which may prevail, such as temporary irritation of the kidneys by drugs or toxins, febrile states, changes in the composition of the blood. The treatment of the albuminuria due to above-mentioned factors depends upon the cause.

Hematuria may be of renal, ureteral, vesical or urethral origin. Renal hematuria in the aged is always a grave symptom indicating either a profound change in the blood or an intense irritation and congestion of the kidney. Ebstein reported a case of an aged patient with hemorrhagic infarct of the kidney and hematuria with rapid recovery, but such cases are rare. The principal causes of renal hemorrhage are nephritis, acute hyperemia, calculus, cancer, papilloma, pyelitis, tuberculosis, infarction, traumatism, infectious diseases, pernicious anemia, leukemia and late cirrhosis of the liver. Ureteral hematuria is generally due to impacted calculus or to the passage of a rough-edged stone. Vesical hematuria is usually due to vesical calculus, acute cystitis, ulcer or growth in the bladder. Hemorrhage from the urethra is generally due to traumatism or enlarged prostate. In renal hematuria the blood is intimately mixed with the urine and is smoky; in vesical hemorrhage the first part of the urine voided is clear, in ureteral hematuria the blood appears in small clots, while urethral hemorrhage pure blood can be pressed out of the urethra.

Hemoglobinuria occurs in cases in which the red blood cells are destroyed by infection, toxins, or drugs, or by diseases like pernicious anemia, leukemia, scurvy, etc.

The urine in hemoglobinuria resembles the urine of hematuria but there are no blood cells in the former.

Hematuria and hemoglobinuria are incidental symptoms occurring in many diseases and while not pathognomonic of any, they have a corroborative value of the greatest importance. In hemoglobinuria the kidneys are usually not affected, in

hematuria of renal origin the kidneys are usually diseased either primarily through local irritation or secondarily through blood changes.

The treatment depends upon the cause. Local treatment is useless in hemoglobinuria as the causes lie in the blood. In renal hematuria benzoate of ammonia in 5-grain doses is sometimes of benefit when due to infectious disease, and camphor in 5-grain doses when due to drug irritation. Astringents are of service in vesical hematuria. The underlying cause must be treated in all cases.

Pyuria occurs in all suppurative conditions of the urinary organs and passages. It will also occur when an abscess in an adjoining tissue breaks into a urinary passage. The most frequent causes in the aged are cystic infection introduced by the catheter and vesical and renal calculus. The recognition of pus in the urine is of importance in the differential diagnosis between senile non-infectious cystitis and chronic infectious cystitis. It may also direct attention to the location of an infection giving constitutional symptoms, but no pronounced local ones.

Other urinary abnormalities are occasionally met with, but they present no marked difference in etiology or diagnostic value, from those of earlier life. *Indican* is usually found in larger quantities than in maturity, while the amount of mucus and calcium salts is diminished.

Acute nephritis is generally due to acute irritation by drugs. Scarlet fever, diphtheria, typhoid fever and other infectious diseases which are the principal etiological factors in early life are very rare in the aged. Exposure to cold and wet, another potent etiological factor in younger individuals, is also less prevalent in the aged. The disease does not differ from the acute nephritis in younger life and requires the same treatment. It occasionally becomes chronic.

Chronic parenchymatous nephritis is rare and when occurring follows the same course and presents the same symptoms as in younger individuals. It is, however, a graver disease from the onset, the symptoms are more pronounced and it passes through the three stages rapidly, death, due to uremia, sometimes occurring within a few months after the acute initial symptoms have appeared. It is rarely prolonged beyond a

year. No plan of treatment has been successful where the degenerative changes due to the active inflammation have been added to the senile degeneration. The plan of giving a salt-free diet cannot be followed in the same manner as in younger individuals, as the sudden withdrawal of salt causes anorexia and inanition with consequent rapid exhaustion. Salt, alcoholics, coffee, etc., to which the patient may have been accustomed for years, must be withdrawn gradually. The exclusive milk diet has the same objection since large quantities must be taken to supply sufficient nutrition. Malted milk is a valuable substitute which will not become objectionable too soon, and will not require such enormous quantities of fluid as ordinary milk does.

Drug treatment is purely symptomatic. Irritant diuretics aggravate the condition of the kidneys. The best diuretic in these cases is a saline, either potassium nitrate, citrate or acetate or lithium citrate. The natural lithia waters contain a large proportion of lime, which is contraindicated in senile cases. The general treatment suggested under chronic interstitial nephritis will apply to this disease.

Pyelitis is rare, most cases being due to the irritation produced by a renal calculus, followed by infection. Typhoid fever, which is a frequent cause of this condition, is rare in the aged and likewise tuberculosis and cancer of the kidney. The disease does not differ from pyelitis in earlier life and must be treated the same way.

Renal and perirenal abscesses and cysts may occur, but the causes are rare and in their pathology, symptomatology and treatment do not differ from the same diseases in maturity.

MYALGIA

Myalgia, or muscular rheumatism, occurs frequently both in the acute and the chronic forms.

Etiology.—The most frequent cause of myalgia is a sudden chilling of the surface especially when it had been overheated. It is probably due to the same toxins that produce fatigue, as it occurs most frequently in muscles which have been subjectd to extraordinary exercise or where, owing to poor surface circulation, a sudden chilling will still further interfere with the

circulation and prevent the removal of the toxins produced by muscle activity. *Myalgia pectoralis*, pleurodynia, occurs frequently as the result of hard coughing or sneezing; *myalgia lumbalis* or lumbago occurs most frequently when the surface had been chilled; *myalgia cervicalis*, torticollis, may be due to a draught or to extreme rotation of the head; *myalgia capitis*, cephalodynia or rheumatism of the scalp, is generally due to exposure when the scalp is warm, as for example when a person in a heated room puts his head out of a window during extremely cold weather. The dull ache of chronic or prolonged myalgia is identical with the pain in muscles that have been excessively exercised and the relief from these pains is brought about by measures best calculated to promote local circulation which would remove these toxins. There is no evidence that the disease is due to a bacterial infection, but causes that interfere with local circulation such as arteriosclerosis, or conditions interfering with the ability of the blood to carry away toxic material, as in the gouty diathesis, autointoxication, etc., predispose to it. There is also probably a neuralgia or neuritis present, the initial symptom generally starting with a sharp neuralgic pain as in sciatica or in intercostal neuralgia, and this pain can be reproduced upon motion.

Pathology.—No distinctive lesions have been found, the usual anatomical changes discoverable upon autopsy in cases where death had occurred from some other disease while a patient had an associated myalgia, being due to senile degeneration of the muscles and nerves.

Symptoms.—The initial symptom is usually a sharp neuralgic pain which is soon followed by a persistent dull ache aggravated upon motion. Some authors say the pain is increased upon pressure; others say pressure lessens the pain. Steady pressure without motion during the neuralgic stage lessens the pain, but during the later stage pressure increases the ache while sudden motion will produce a sudden sharp pain resembling that of neuritis. The pain in the muscle may extend to the tendons and aponeuroses. In some cases the initial pain lasts but a moment or may be absent altogether, in others there may be paroxysmal attacks even without motion. The dull myalgic ache is always present while the sharp neuralgic or neuritic pain is occasionally absent. The disease is usually

unilateral, rarely extending beyond one muscle or group of muscles. The affected part is always held in a position causing the least strain upon the affected muscles, shielding them as far as possible from motion. While neuralgia is an important element in the diagnosis and treatment of myalgia, the recognition of myositis is more important. Neuralgia is of short duration; there is a painful point along the nerve, while the surrounding tissue is not painful. In myalgia there is no painful point unless the accompanying neuralgia is persistent and severe; there is local tenderness, however, and the disease is prolonged. There should be no difficulty in differentiating between myalgia, myositis, pleurisy, spondylitis, costal caries, cancer, renal calculi, etc., which all give localized pain. The aches due to senile waste of muscle are increased upon motion, but there is no pain when the patient changes his position in bed.

Treatment.—The treatment of myalgia consists of rest, heat and the avoidance of the cause. In pleurodynia it may be necessary to strap the affected side with strips of adhesive plaster to secure the necessary rest for the affected muscles. Heat should be applied in the shape of hot poultices, turpentine stupes or cataplasms. An inunction of equal parts of chloral and camphor or menthol and camphor will relieve the pain of the neuralgia and may relieve the myositis. Extremely hot baths, as the Turkish or Russian baths, are dangerous in old age. If the pain persists and is severe, it may be necessary to use hypodermics of morphine and atropin, but other internal medication is useless. The causative condition, if due to gout, autointoxication, or similar endogenous factors, should be treated.

Myositis, inflammation of muscles, is rare. A chronic fibrous myositis may occur as a manifestation of tertiary syphilis, while acute primary myositis is believed to be due to infection or fatigue toxins. The chronic form is an interstitial myositis, with proliferation of connective tissue and atrophy of the muscular fibers, the muscle mass appearing in some parts swollen, in other parts atrophied, in parts soft, in others firm. The disease spreads over large areas or is scattered over many muscles, there is a dull ache worse on motion or pressure and sometimes worse at night. The symptoms are relieved by antisyphilitic treatment. In the acute form there is rapid atrophy of the affected muscles partly through degeneration and partly

through pressure of round-celled proliferation of the connective tissue. There is a gradually increasing pain in the muscles and progressive loss of power. The affected muscles are at first firm and apparently swollen, later they become thin and soft. The disease resembles *myalgia*, but the increasing pain, atrophy and loss of power should serve to distinguish it from the other. In rare cases there is fever and swelling and the disease resembles *acute articular rheumatism*, there is, however, no pain in the joint itself, while the muscle pain is constantly increasing, and muscles in other locations than over joints are also affected.

Trichinosis gives symptoms similar to acute primary myositis.

Examination of the blood shows an increase of eosinophiles, but a positive diagnosis can be made only by examining a piece of muscle under the microscope. In *polyneuritis* there are painful points or the pain is along the line of the nerve, the surface is tender and there is no atrophy of muscle. No treatment is known for myositis. The salicylates are of service if there is fever and narcotics may be required for the pain. The disease occasionally disappears without treatment.

Progressive muscular atrophy is almost always the spinal form, which had been carried over as a very slowly progressing disease from maturity; it never originates in old age. The history and the symmetrical atrophy without sensory disturbance, diminished tendon reflex, fibrillary twitching, and absence of disturbance in organs of spinal origin distinguish it from other diseases in which muscular atrophy is a prominent symptom. The disease is incurable and slowly progressive, and nothing is known to retard it.

MÉNIÈRE'S SYMPTOM COMPLEX

Ménière's disease of the labyrinth is extremely rare but the symptom complex with some modifications is of frequent occurrence in the aged. When due to other causes than disease of the labyrinth it is known as pseudo-mèniére's disease.

Etiology.—In a few cases the symptom complex is due to middle or inner ear affections. More often it follows a general disease such as syphilis, gout, diabetes, leukemia, general paresis, or may follow injury to the head or disease of the nose. It

may also occur in hysteria, neurasthenia and psychic disorders. The most frequent cause is arteriosclerosis of the vessels of the ear or brain.

Symptoms.—The symptom complex consists of a paroxysmal rotary vertigo, generally so severe as to produce momentary unconsciousness, followed by headache and usually nausea and vomiting. Tinnitus and difficulty in hearing may precede but generally follow an attack. The vertigo generally lasts a few minutes, rarely over a quarter of an hour, but the headache and nausea may last for several hours, while the tinnitus and deafness may be permanent but varying in degree. If the vertigo is prolonged, ataxic symptoms appear, but these disappear as soon as vertigo passes away. The ear symptoms may appear on one side or on both sides. In some cases there is a persistent mild vertigo with sudden exacerbations, in other cases complete deafness occurs and all other symptoms disappear.

Treatment.—The treatment depends upon the underlying condition. There is no known cure for the labyrinthine disease, but the symptoms usually disappear as soon as complete deafness occurs. When due to other causes the cure of the primary condition will relieve the symptom complex. Large doses of the bromides will usually relieve the nausea, headache and other secondary reflex symptoms following the vertigo. The only relief of the vertigo is found in the recumbent position with the head low.

OSTEOMALACIA

Etiology.—**Osteomalacia** is probably due to some perversion in the function of the thyroid gland. The relation of the thyroid to metabolism is still somewhat uncertain and there seems to be a tendency to ascribe all trophic changes of unknown origin to thyroid disease. Osteomalacia has, however, been found most prevalent where goiter is endemic, and Grajon states that it is frequently found in the aged insane.

Pathology.—The anatomical changes are a waste of cancellous structure and a resorption of lime salts, later the harder structure about the Haversian canals also soften. The periosteum generally becomes thin and sometimes separates from the bone; in rare cases it becomes thicker. The medullary canal is increased in diameter and the marrow is at first red, later it

becomes yellowish and gelatinous. The most marked changes are found in the spinal column.

Symptoms.—The disease in the aged begins with persistent slight aches and pains, generally in the back and loins, sometimes in the extremities, and with increasing difficulty in motion. As the disease progresses the pains become more severe and motion is thereby restricted until finally the patient is confined to his bed avoiding the least possible change of position. Owing to the pain, the aged patient does not walk or stand much and the deformity in the lower limbs does not become as marked as in Paget's disease, but as he sits more there is a greater curvature of the spine consisting of both a scoliosis and a kyphosis with consequent malformation of the chest walls. This change and the compression of the pelvis, depression of the neck of the femur and curvature of the long bones of the lower extremities produce a marked diminution in a stature. In some cases the ribs reach the ilia. The least affected are the bones of the skull. As the disease advances mental depression ensues, the face becomes dull and expressionless. Later constitutional symptoms will appear such as anemia, antointoxication following constipation dyspnea and cyanosis, then cardiac and circulatory disturbance with trophic disorders in all organs and tissues. The diagnosis is difficult at an early stage of the disease and can only be made by excluding chronic rheumatism, gout and other arthritic diseases, syphilis, tumors, carcinoma, various neuralgias, tabes and other spinal affections. Syphilis is excluded by nocturnal exacerbations of pain by the history and by the result of treatment; rheumatism by its location in the joints; gout by the location and character of the pain. In these and other arthritic diseases the impairment of motion is due to a stiffening of the joint, while in osteomalacia impairment of motion is due to weakening of the bone. Tumors give localized symptoms and cancer can usually be diagnosed by the history, the presence of cancer in other localities and the local and general symptoms. The neuralgias are localized and the pains are paroxysmal. Tabes has pathognomonic symptoms and signs and in other spinal diseases the pains are not diffuse, nor is there bone tenderness. Paget's disease is more localized, thse is a hyperplasia of bone, and pains, if present, are not severe.

Treatment.—Phosphorus is virtually a specific in osteomalacia. The rationale of this treatment lies in the property of phosphorus to combine with lime to form phosphate of lime which is deposted in the bones. The dose is 1/100 grain three times a day. It can also be given in the form of glycerophosphate of lime, but lime salts not in a phosphorus combination are not taken up in this disease. Dietetic and hygienic rules must be observed. Sulphur baths and massage may be employed, but exercise or any active motion is prohibited. Codliver oil has done good in some cases.

Osteomyelitis is rare in the aged. Fatigue is believed to be an important etiological factor in senile cases and when it attacks an aged person it is generally after excessive walking, when the tibia will be found affected. In some cases no cause can be found. The symptoms are pronounced, the pain is intense and increasing in severity and the constitutional symptoms of infection are grave. In a few cases mild symptoms prevail but the disease in the aged is usually fatal.

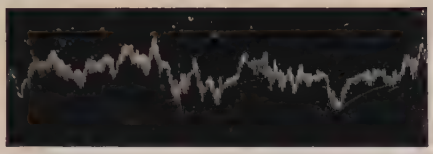
Treatment is the same as in earlier life, but in the grave form operation alone offers any chance of recovery.

SPINAL DISEASES

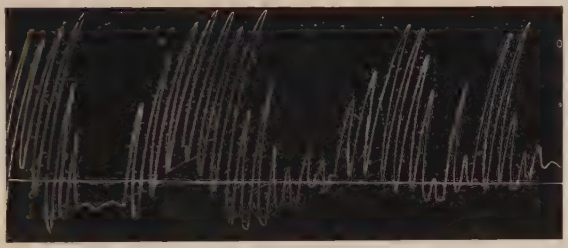
Acute myelitis is rare in the aged, the principal etiological factors, toxins and infectious diseases being of infrequent occurrence. It does not differ from the acute myelitis of earlier life, presenting the same lesions and symptoms. It has a greater tendency though to become chronic.

Chronic myelitis following the acute attacks is of short duration. In these cases vesical and intestinal paresis generally follow with consequent autointoxications, sepsis and exhaustion. This form of chronic myelitis differs from the senile myelitis in which there is a slow, progressive, but never complete, paraplegia, and the intestines and bladder are but slightly if at all involved. Treatment is the same as in earlier life.

Compression myelitis is almost always due to veretbral caries or carcinoma. A very rare cause which does not prevail in earlier life is the compression produced by a beady hardening of the vertebral artery in arteriosclerosis of that vessel. The symptoms are the same as in maturity and depend upon the location of the lesion. The causes being persisting and in-



Tremorgraph—Multiple sclerosis. (Neustaedter, *Med. Record*, July 17, 1909.)



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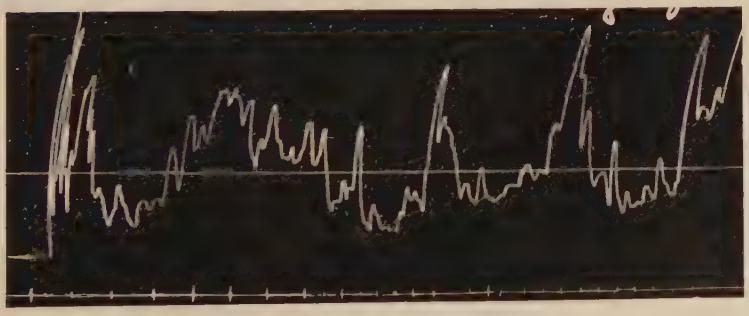

Tremorgraph—Dementia paralytica. (Neustaedter, *Med. Record*, July 17, 1909.)

creasing in force or extent, the symptoms also persist and usually become more intense or involve larger areas, and there are no remissions. The compression myelitis due to caries is slowly progressive and there is a dull ache. When due to cancer it is rapidly progressive with intense pain.

The treatment depends upon the cause. Orthopedic appliances are indicated in cases of caries without abscess. Tuberculosis or syphilis when present must receive appropriate treatment. Cancer being usually a secondary condition following cancer in other tissues, nothing can be done except to relieve the symptoms. It may be necessary to resort to narcotics in such cases. Spinal diseases generally are rare in the aged and many degenerative changes which in earlier life give pronounced symptoms are found upon autopsy of aged persons, to have produced no symptoms of spinal affection during life. Clearly defined symptoms of tabes dorsalis, spastic spinal paralysis, multiple sclerosis, syringomyelis, etc., are very rarely observed after the sixtieth year, while the lesions themselves are not at all infrequent. The lesions of multiple sclerosis of the cord are frequently found after death and appear to be nothing more than the normal senile changes. It is only when these degenerative changes occur as a result of abnormal factors, such as syphilis, infectious disease, growths or traumatism that they present pronounced morbid symptoms. In senile cases the symptoms are generally milder and more prolonged than in maturity but pain is more intense, while muscle rigidity and motor paralysis may proceed to complete loss of motor function.

Cases of spinal disease may be carried over as chronic affections from earlier life and, owing to more rest and better hygienic environment enjoyed by the aged, the symptoms may become milder. The degenerations, however, cannot be repaired and are usually progressive. The treatment of spinal affections in the aged is the same as in younger individuals, care being taken in the selection of drugs to guard against secondary effects upon the heart and blood-vessels. This applies with special force to strychnine.

CEREBRAL DISEASES

Meningitis is very rare in the aged. *Pachymeningitis interna* may occur in cerebral atrophy but its symptoms are

not clear. Paroxysmal attacks of headache, temporary attacks of hemiplegia, unilateral muscle cramps, and temperamental changes have been found in connection with it, but there is no pathognomonic sign or symptom complex.

Purulent meningitis may occur with mastoiditis, otitis media, erysipelas and other infectious diseases. It begins with malaise, violent headache and other cerebral symptoms pointing to meningitis. Rigidity of the muscles of the neck and extremities occurs. The cranial nerves in the locality of the inflammation become involved and the functions of the parts supplied by them, are impaired. Kernig's sign is present. The reflexes are first exaggerated, later abolished. The presence of pus in the cerebrospinal fluid confirms the diagnosis. The ordinary treatment for meningitis, rest, quiet, ice bags to the head, narcotics, etc., apply to this condition. Serum therapy may be tried. Lumbar puncture gives temporary relief.

Tubercular meningitis may occur in connection with pulmonary tuberculosis. The symptoms are the same as in purulent meningitis and the same local treatment is indicated. The underlying disease needs attention. Meningeal affections in the aged are usually secondary, run a chronic course and while incurable death in most cases is due to the underlying disease.

Syphilis of the brain is very rare after the sixtieth year and unless there are gummata which produce cerebral compression, there are no clearly defined symptoms.

Abscess of the brain is generally due to traumatism, occasionally to pyemia, rarely to infectious peritonitis, endocarditis or other infectious inflammation or to abscess or gangrene elsewhere. It does not differ from the same condition in earlier life.

HOME CARE OF THE AGED

The old man wants constant attention and needs constant care. When he begins to feel the weight of his limbs and the creaking of his joints, the growing weakness and the loss of virility, when he begins to notice the hundred and one indications that betoken the advancing years, his whole being becomes wrapt up in himself. His thoughts turn toward death and his one aim is the preservation of life. From this moment he becomes an object for his solicitude. While the mind is still bright he notes the little aches and pains that accompany old age, and avoids every motion that might aggravate them, thereby losing the benefit of exercise. As the sense of taste diminishes, he wants sharper and more spicy food and with the loss of teeth he swallows the food in lumps. He avoids straining at stool and becomes constipated, the feces remaining in the colonic pouch. He finds a little difficulty in voiding urine and he defers the act until the accumulated amount gives him distress and in the meantime dilatation of the bladder is produced or increased.

When the mind becomes impaired he neglects his person in every direction until he becomes obnoxious to those around him. He cannot accommodate himself to a progressive order or to modern ideas, he becomes old fashioned, even queer, while those nearest to him try to humor his whims until patience is well-nigh exhausted. At the same time he demands constant attention and complains of the slightest neglect. The firm insistance upon hygienic measures for his benefit and welfare, which necessarily impose some exertion on his part, is resented as a hardship and creates a dislike of those who are most interested in his welfare. This is the foundation of oikiomania, the morbid state in which the natural love for those entitled to the love of the individual, is turned to hatred, without reasonable cause. As this is common among the aged who live with their family, it is the principal bar to the successful treatment of

diseases and the proper hygienic care of the aged at home. Rather than force the old man to take proper care of himself, and thereby incur his displeasure, they submit to his whims and permit him to deteriorate mentally and physically faster than he would under other circumstances.

In many cases it is possible to overcome the prejudices of the aged by tact, and to create in them a sense of well-being. In the article on Senile Cachexia it was pointed out that many cases of decrepitude were really cases of pseudo- or psychic debility and that the removal of certain factors causing this pseudo-debility would rejuvenate the aged.

Mental stimulation is the most important measure in the hygiene of the aged. Anything which will tend to make the senescent take an interest in life beyond their own little ego will benefit them. We may repeat here that the psychic influence of flattery is more potent in arousing ambition than drugs or reasoning. It will arouse renewed pride in appearance which is usually lost when ambition is lost in the contemplation of death in the near future.

Just as long as this pride in appearance is maintained, so long will the individual follow willingly the hygienic rules necessary for his welfare, even though it requires some exertion and effort to carry them out. Mental activity arouses physical activity and creates vigor if the organism is still in the condition to respond. The most powerful of the mental stimulants are, change of scene and residence, change in the mode of living, a young wife or husband, discussion upon some familiar favorite subject, or a hobby. The joke about the bald heads in the theater where there are pretty chorus girls has a psychological basis. Mental activity is aroused and the old man feels young again. Whatever the means may be, the end to be attained is the same, therefore, mental activity must be encouraged, and pride in personal appearance stimulated. If this much is accomplished there will be little difficulty in dealing with the old.

The other principal hygienic measures are food and exercise. Cleanliness and clothing are secondary, for notwithstanding the importance of cleanliness from an esthetic point of view, many reach advanced old age who have rarely taken a bath and who have had little choice in the selection of clothing. Fresh air is necessary and the air of the country is better than the air of the

city. Caution as to the evacuations seems superfluous, as this applies to all ages and to all conditions. Still the aged pay less attention to the stools and urine, especially if there is any difficulty in passing either, and this neglect causes pathological conditions. They should go to the toilet at the same hour each day and the family should not place too much reliance upon the patient's assurance that it is "all right." The family should attend to the cleansing of the catheter and the rectal tube if these are used, and they should be made sterile again just before they are to be employed. A daily bath entails too much exertion for the old man who has no attendant to help him in and out of the tub. A tepid bath once a week will accomplish all that is necessary in the case of the aged, but daily ablutions of the hands, face and neck should be insisted upon. The common laundry soap should not be used as it makes the skin excessively dry. Either a mild soap like castile soap or plain borax can be employed, and sea salt or common salt should be used for the bath. For the bromidrosis, washing with Florida water or cologne water and powdering the part with a mixture of stearate of zinc and salicylic acid will generally relieve, and may cure, this condition. As the surface temperature of the body is generally low and the aged do not perspire readily they should wear warm woolens all the year round. In winter heavy underwear will keep them warmer than a heavy overcoat and there is less weight to carry. The legs and feet should be especially looked after as the lessened surface sensibility makes the aged less sensitive to temperature changes, while the poorer surface circulation in the lower limbs makes them especially liable to chilblains, frost bites and frozen feet and toes. This is a frequent cause of senile gangrene. The same precaution need not be taken with the face but in very cold weather the ears should be protected. An important article of wear is the shoe, which should be selected with due regard for corns, bunions, hammer-toes and broken-down arches. It should have rubber heels and arch supporters whether there are broken-down arches or not. Apparently insignificant, yet really of great importance, is the cane, upon which the old man depends for support when the senile kyphosis and waste of the muscles of the back make him stoop. With the cane he is able to maintain a fairly erect position and if he

uses it as soon as he begins to notice the tendency to stoop or to fall into the attitude of the senile slouch, he will keep erect and lessen the strain upon the back muscles and the compression of the intervertebral discs. The cane should be sufficiently long so that when the point is on the ground at the distance of the ordinary step from the body, the user will not be obliged to stoop over when grasping the handle. Most canes are too short and the man must do the very thing the cane is intended to prevent.

The diet of the aged must be regulated by the state of the teeth, stomach and intestines, and by the metabolic activity and assimilation. The senile organism requires less food, it can dispose of less food, and less is assimilated. Owing to the changes in the stomach, digestion is slower and weaker. With bad teeth the food is not properly masticated, consequently it is swallowed in lumps, which are digested with difficulty or not at all. Prosthesis can remedy this defect, and here again it is often necessary to play upon his pride in appearance to induce him to undergo the annoyance and sometimes positive distress connected with the making and wearing of artificial teeth. Women submit to these discomforts more readily than men, for their natural vanity induces them to appear attractive, even when extremely old. Gourmands who are accustomed to eat too much will not break themselves of the habit when they become old unless gastritis and diarrhea make a limited diet imperative. The oft-repeated advice that the aged should eat little and often is irrational, for digestion is naturally slower in old age and frequent feedings keep the stomach constantly at work, there being always a mass of food in the stomach in different stages of digestion. This is the most frequent cause of flatulence, heart burn and senile gastric catarrh with its attendant pyrosis and gastrodynia.

In old age, food should be taken not oftener than once in five- or six-hour intervals at fixed hours each day. The number of meals, like the time of day when the principal meal is taken, is a matter of habit, often of nationality, and does not affect the rule. A few simple directions will serve better than any fixed diet list.

If food cannot be masticated it should be chopped up fine or administered in the form of mush. No food should be taken

between meals. Milk, buttermilk, weak tea, coffee, strained cocoa, can be taken, however, preferably an hour after meals. Meat should be used sparingly, not oftener than once a day, preferably underdone. Pork is forbidden. Fish and shell fish may be taken if they do not produce ill effects but if they harm once, they will harm again. Vegetables containing much fiber like cabbage, turnips, carrots, sweet potatoes, etc., leave a large amount of waste and induce peristalsis. The cereals and the breakfast foods are all good. "Wine is the milk of the aged." Light wines like Hock, Moselle, Claret, Burgundy, etc., are the best. Port, old Sherry and Madeira wines contain too much alcohol. Beer and ale may be taken if they do not produce flatulence or pyrosis. The day should be begun with a glass of hot water containing a little table salt or if constipation exists a teaspoonful of any of the cathartic salts, and at night a glass of hot milk may be taken before going to bed. As the aged person is liable to awake during the night a glass of milk can be left at the bedside. Gastric and intestinal disorders may necessitate modification of these rules. A safe general rule is to avoid all purin-forming substances, foods containing much cellulose and foods containing a large percentage of water. This will hold good for all conditions. Notwithstanding all that has been said and written against drinking and smoking most men who have reached advanced age have indulged in both. Everything in excess is harmful. As for the determination of what is excessive every man is his own judge. When a man has lived so rational a life that he has reached old age it can be safely left to his own judgment to decide how much he can drink and smoke without harm.

The question of exercise is intimately bound up with mental stimulation. The aged need mental and physical exercise and recreation, the form of both depending upon the mental and physical condition of the individual and the proper application of the rule that recreation should be the antithesis of the work necessitating it. Mental labor, requires physical recreation, a sedentary occupation requires activity, etc. The same holds good for the aged but as all forms of activity are diminished and fatigue sets in more rapidly, exercise and recreation must be milder than in maturity. Physical activity cannot be prolonged on account of the weakened locomotory tissues, these

soon becoming tired, also on account of the increased action of the heart and lungs which cannot keep up prolonged hyperactivity without increasing their own degeneration. Still, some form of exercise is necessary to prevent stiffening of the joints. It is hardly in place here to discuss the theoretical necessity of exercise to produce heat and increase metabolic activity. Joint motion must be undertaken to prevent anchylosis, even though it increases waste which is not repaired. The best form of exercise for the aged is walking up a slight incline with frequent rests. This exercises the muscles of the lower extremities and of the back and if a cane is used, the muscles of the upper extremities are also brought into play. A walk through an unfamiliar forest path will not alone give physical exercise but will stimulate the brain and cause continual mental exhilaration. Nothing, however, equals a few hours of fishing when fishing is good.

Active athletics are naturally out of the question, even gymnastics cannot be undertaken, but calisthenics are beneficial. An imperative rule in all forms of exercise in the aged is to stop the moment fatigue sets in or dyspnea or palpitation is produced. Mental stimulation cannot be continued beyond its physiological limit, for when brain fatigue sets in, the aged individual falls asleep. This is seen in the case of the old man who falls asleep during the sermon. It is not lack of attention but prolonged mental concentration that causes the brain fatigue and sleep. This should be understood by speakers who resent the seeming slight when an old person falls asleep during a sermon or lecture. Even sensory stimulation can produce mental fatigue, as is seen when the aged fall asleep at the concert or spectacular play. Mental stimulation should be agreeable, otherwise it is mental irritation which is depressing. Discord in music, the whirl of the dance, the shouting at a game irritate, while melody, the harmonious movements of the ballet, catchy songs sung by a chorus, stimulate and create cheerfulness. The play which demands constant attention to understand the slow unraveling of the plot is wasted upon the old man, and also the play which is complicated or where the action is so rapid that the mind cannot follow it. The selection of the play, concert or similar diversion depends upon individual taste, but the mental capacity should not be

overlooked. The outing is an agreeable form of diversion, especially if young people take part and do not neglect the aged. If the old man likes fishing and hunting he may indulge in these pastimes, but rowing is too strenuous.

Travel or a change of scene has often a wonderful effect upon the mind of the aged. If accustomed to the lowlands or seashore it is dangerous to take him to high altitudes. With this precaution, the destination should be a place in which he is interested and which he has not seen before, or not in years. The object should always be, to arouse in him an interest in something else than his body. A favorite pastime of old people consists in reading old familiar books, and in gossip. The old man's gossip is mostly reminiscences, the old woman's does not differ from the gossip of her younger days.

The woman shows herein the greater interest in life, for she is interested in the doings of her sisters while the old man's talk begins with "I" and ends with "me." But even that is better than the reading of old books, because he has listeners who in turn tell their tales of "I" and "me" and so create new interests.

There are many little factors in and about the house which can be included in the hygiene of old age. The old man should have an easy chair with padded arm rests. Without such rests his hands lie in his lap and his shoulders fall closer together.

With his arms on the rests his shoulders are thrown back, the upper part of his chest is expanded giving more room for the expansion of the lungs and he breathes deeper and more freely. If the chair is slightly tilted when he takes his nap his head will fall backward and he will snore. If his head falls forward the vessels of the neck are compressed, which produces a passive cerebral hyperemia, as is evidenced by the flushed face and injected conjunctivæ when he awakes. Let the old man snore, but if he groans while sleeping with his head upon his chest, he should be awakened. Deafness and presbyopia are common ailments and the former, especially, causes mental depression and may lead to oikiomania and melancholia. The old are selfish and suspicious, they feel they are practically useless, that they are a burden upon those who look after them. When they cannot hear what is said around them, a glance in their direction is sufficient to arouse in them the suspicion that

they are the subject of a conversation held in their presence, and such suspicions invariably lead to perverted conclusions. To avoid this, as soon as it is noticed that an aged person makes an effort to hear what is said, that he turns one ear to the speaker or watches the movements of the lips, or gives other evidence that hearing is becoming impaired, he should be furnished with a speaking tube or other appliance to improve his hearing.

Drugs are useless. If the sight is impaired the nature of the impairment should be ascertained. In most cases it is simply a presbyopia which can be remedied by proper glasses. It may be cataract or a progressive amaurosis probably due to senile degeneration of the optic nerve.

The aged are grateful for little attentions, such as an occasional nosegay, but if given for several days in succession they expect them and a single neglect to furnish them is cause for complaint. The memory of such neglect is hoarded and brooded over for days. If the old man or woman has a harmless hobby which is not silly and will not expose the individual to ridicule or interference, it should be encouraged. Unfortunately the hobbies of the aged are not always harmless, they are often childish, sometimes insane. It is extremely difficult to break an old person of a new hobby, especially if it involves sexual perversion or other moral defect. As the moral sense becomes blunted he cannot be made to realize the wrong in his actions and it may become necessary to instil fear of punishment to hold him in check. As the mental and physical powers wane, the aged find comfort in the association with children, especially in the companionship of a favorite grandchild or niece. Much can be done with them through the influence of such favorite child, and such companionship should be fostered. The widespread belief that the aged regain youthful vigor at the expense of the child has nothing to uphold it.

The family should be taught to observe slight changes in the physical condition and demeanor of the aged under their care. The symptoms of disease begin so insidiously and progress so mildly that a grave disease may be far advanced before the family realizes that the patient is ill. The aged seldom complain of pain or give other marked symptoms of disease, the mind and the perception of pain being blunted and it frequently happens that the earliest manifestation of the

disease is collapse. When an aged individual who is accustomed to be up and around shows a disinclination to leave his bed, it indicates a rapidly growing weakness such as accompanies senile pneumonia. The family says he is failing rapidly while it may be the exhaustion which accompanies a grave, probably a fatal, disease. When the aged individual talks in his sleep, and has never before done this, we have probably a low muttering delirium indicating a cerebral disorder. The family should learn that a cool forehead may exist with high fever, that surface temperature is no indication of body temperature. A chill is always a signal of danger requiring immediate attention. Vomiting after a heavy meal is often the first sign of acute gastritis which is always a grave disease in the aged. If he is too long in the toilet he may have fallen asleep or he may be straining to relieve a distended bladder which is blocked by a calculus or a hypertrophied prostate. A cold sweat on a pale face is a grave symptom generally indicating collapse. There are many causes for coma but when occurring in old age only that of apoplexy and embolism is sudden and without antecedent recognized chronic disease. If the face is flushed, the head should be raised and ice bags applied. If the face is pale, hot applications should be placed upon the head while the patient lies on his back with the head low. The same rule holds good if there is headache. Anorexia is not a dangerous symptom by itself and is frequently due to gastric catarrh. If there are other symptoms not pointing to gastric disorder, there is probably some serious disease present, anorexia being one of the earliest symptoms of inflammatory conditions. Sudden irritability indicates either mental disorder or distress, perhaps not amounting to pain. If the patient complains of pain anywhere it should receive immediate attention. Occasionally the aged will complain of pain to arouse sympathy. It is difficult to detect such malingerers, especially if they refer the pain to some internal organ. Repeated examinations may be necessary, but the individual generally betrays himself by forgetting the spot where he located the pain at the former examination.

INSTITUTIONAL CARE OF THE AGED

What has been said in the preceding chapter applies to a great extent to the care of the aged in asylums. There is,

however, a vast difference between the asylum and the home, and between asylums among themselves, and the care bestowed upon their inmates. There are private asylums, sectarian and unsectarian, to which a large admission fee is paid, private asylums maintained by organizations for their members who have contributed toward them and hence have a proprietary right to them, semi-private asylums maintained by nationalities, churches and vocations for those of the same nationality, church or vocation, and public asylums or poor-houses. The care bestowed upon the inmates naturally depends upon the class of institution. Those who pay large admission fees belong to a strata of society in which refinement in surroundings is imperative, luxuries are necessities and the utmost care is expected. At the other extremity is the poor-house, the inmates of which are paupers from the slums of the city and the poorest inhabitants of the country. One fundamental difference between the aged at home and in the asylum is in the mental attitude. In the asylum there is freedom from care about the future, from worry about the family to whom the individual had probably been a burden, and from fear that the family is trying to get rid of him and might go to extreme measures to secure relief from the incubus. There is on the other hand the feeling of dependence and a sense of lost independence, restrictions in many directions, in actions, in food, perhaps in clothes, the inmate must obey rules, perform tasks, and above all he must not complain. In the public asylums there is a sense of absolute helplessness, the inmate feels that he is dependent upon the bounty of every individual in the community, that complaint will be followed by punishment, that he is virtually a beggar without rights. Under such circumstances the inmates of alms-houses become morose, apathetic, they lose interest in everything except themselves, and melancholia and senile dementia follow. It is impossible to arouse in them any sense of pride in appearance, any ambition, or interest in anything.

In New York City the name alms-house has been changed to City Home for the Aged and Infirm. This has had an elevating influence upon the inmates who are now no longer paupers of the alms-house, but inmates of the city home. The establishment of the city farms for them has had a further beneficial

effect in stimulating interest—the great desideratum in dealing with the aged. In a great public institution intended for all races, religions and nationalities, the inmates form sets and cliques based upon similarity in race, religion or nationality and this gives rise to jealousies and ill-will. The inmates being drawn mainly from the lowest strata of society, they comprise the quizzical and querulous, the shrinking and the defying, the meek and the humble, and the dominating spirits found in the slum sections of the cities, and harshness is often necessary to enforce order among them. With such characters kindness is construed into weakness and it requires tact, patience and firmness to prevent excesses, especially if crippled and aged are thrown promiscuously together. The leaders in complaints and demands are generally the cripples who, being mentally brighter than the aged succeed in securing better treatment, often better food than the others. In institutions where the aged have light tasks assigned to them they do not break down mentally either as soon or as completely as where the aged have nothing to do but sit on a bench and brood. In the large state institution at Lainz near Vienna, which the author visited, the inmates receive counters representing money which can be exchanged at a canteen on the grounds for beer, tobacco and other little luxuries. In this way they receive a few cents each day and a certain amount of beer or tobacco. To prevent the ennui which leads to melancholia, the inmates follow their vocations in the institution, as far as they are able, and go twice daily to the canteen which is fitted up as a "bier stube." They have a band, and in other ways their interest in life is maintained. They are naturally under restrictions, but they are at liberty to go and come at will within certain hours, and the depressing idea that they are paupers is not forced upon them.

The care of the aged in public institutions depends as much upon the intelligence, tact and humanity of the person in charge as upon the funds at his disposal. It is naturally impossible to give individual attention to each inmate where there are many, but it is possible to stimulate individual interest in each one's welfare. It is likewise possible to make the inmates more cheerful, rouse them out of the lethargy into which newcomers soon sink, and prolong their lives. The aged like attention,

but they do not like the attention of the sightseer who views them as curiosities. Neither do they want the patronizing and pitying expressions of sympathy from the philanthropists who give nothing else but sympathy. These two classes should be excluded from the public institutions. Inmates who do no work need no recreation and they do not want any. Work, however, stimulates the mind and body and recreation is then appreciated. The women should look after their rooms and be permitted to do such work as they are able to do in the kitchen, and they should be given the opportunity to do needle work which can be sold and part of the proceeds be turned over to them. Even the pauper in the alms-house feels that he is not absolutely worthless if he can do something and receive pay for his work. It need not be much, a few cents a day which the individual can call his or her own, will suffice. The sense of proprietorship if only of a few cents arouses the self-respect that is crushed under the depressing feeling that one is a pauper. This was demonstrated to the author in his visit to the Austrian institution.

The medical care of the aged in the public institutions should receive the same attention as in the public hospitals. In an asylum in New York city the windows of the dormitory were left open all day, while the bedding was turned back over the foot of the bed to permit both the bed and bedding to be thoroughly aired. At night the windows were closed, the heat turned on, and the bedding properly arranged for the night. The beds were cold when the inmates retired and the old people were chilled. Those that had bronchitis at once gave evidence of it and their coughing kept those awake who had no such disease. It is impossible to sleep in a cold bed until the heat of the body has warmed the bed sufficiently to keep the sleeper comfortable. The aged have a lower surface temperature, radiation is less active and it takes much longer to warm the bed by the body of an old person than it does by that of a younger and more active individual. This contributed to keep the old people in the institution awake for several hours after they went to bed, and could have been avoided by warming the bed before they occupied it.

Social intercourse between the sexes should be permitted. To keep them apart, as is usually done, deprives them of one

of the main sources of pleasure that they had before entering the institution. No good reason has ever been given why they should be kept apart. Even in semi-private and private homes this segregation is maintained, yet in some places where this barrier is removed the inmates form a large family party and greater interest is shown in the home and in each other.

Too much stress cannot be laid upon the necessity for mental and physical employment in all classes of institutions, and for both sexes. There should be some system in the distribution of labor, and the work must be of such a nature that sudden and prolonged intermission will not destroy it, and another person can take it up where one drops it. The aged delight in completed tasks and they are stimulated to further efforts. The work therefore should be light and of such a character that it can be completed in a few days or weeks; it should offer a variation so as not to become monotonous, and there should be no element of danger connected with it. Farm work is hard yet there are many light tasks about the farm which meet all the requirements of physical labor for the aged. Gardening, especially the care of potted plants, is an agreeable occupation for the aged, and a little commendation for their work incites them to continued efforts. It is, however, not advisable to create rivalry among the inmates of a home in tasks the outcome of which is beyond their control, as in the growing of plants.

The recreations of the inmates of homes depend upon their mental and physical capacity and the character of the work which requires recreation. The depressing influence of the public alms-house causes rapid mental and physical deterioration and the inmates seek few recreations. They should be supplied with work and diversions. Those who can play a musical instrument should be given the opportunity to do so. There is no better collective recreation than an orchestra composed of inmates and concerts given by them. Dancing and athletics are dangerous, but social parties, masquerades, outings, etc., are harmless and agreeable. Such diversions involve little expense, yet this little is given grudgingly or not at all by communities that see in the aged paupers only economically worthless burdens.

The favorite pastime of the aged is gossip. This does no harm. When they take up reading it is either something of

a religious character or some favorite work that they had read and re-read over and over. They picture anew the scenes described and live again in the world of yesterday. Even this is better than no reading at all, although it does not arouse the same mental activity as a new book. There will always be found some inmates of homes who keep up their interest in the world of to-day, in the passing events, new books, art, and science. Such inmates should receive every opportunity to improve their minds. The newspapers and popular magazines are better than novels, as they do not require prolonged and concentrated interest. Card playing is a simple pastime, the simpler games which require no mental effort being extremely popular in institutions where this pastime is permitted. Lotto, checkers and the various home games in which the chance fall of the dice determines the issue, all keep the mind engaged without involving strain or prolonged attention.

In public institutions individual likes and dislikes are disregarded. It is naturally impossible to conform to the desires of each inmate, but in many instances concessions can be made, especially in relation to food, that may lengthen the life of the individual and make him happier. A Jewish inmate of a public (non-sectarian) institution would not eat certain articles of food proscribed by his faith and as he could get no other food, he became weakened from insufficient nutrition. He was removed to an institution of his own faith and rapidly gained in weight and strength. The remarkable showing of the Jewish homes for the aged is probably due to the greater care bestowed upon the food, especially upon the meats. (Longevity among Jews in spite of unsanitary surroundings is believed to be due to their sobriety and sanitary regulations regarding food.) It is impossible to arrange a diet list which would be generally applicable to all classes of institutions or even to all the inmates of one class. The general dietetic rules given in the last chapter will apply here, but there are naturally many exceptions. A different diet is necessary for those who have no teeth from the diet of those who can chew their food. As constipation is a common complaint among the aged, foods having this tendency should be avoided. These include fresh bread, eggs, liver, pork, rice pudding, sago pudding, milk, nuts, cheese and preserved (salt, potted or smoked) meats and fish.

A pernicious practice which the author found in vogue in one institution was the addition of a cathartic to some article of food once a week. Drugs should not be given indiscriminately, but each case should receive individual care. Bladder and intestinal troubles are common among the inmates of institutions and they are generally due to neglect, occasionally to inadequate toilet arrangements. An apparently insignificant omission in one institution caused the inmates much annoyance. They were not permitted to go barefoot, there was no carpet on the floor of the hall and the toilet was at one corner. Several of the inmates were suffering from dilatation of the bladder and walking in their shoes on the bare floor at night disturbed the whole dormitory. A strip of carpet removed this source of insomnia. Where a large number of aged individuals are collected, daily baths, either tub or shower, become necessary. Constant vigilance is required to prevent an invasion of parasites, for once they gain a foothold it may become necessary to quarantine the whole institution, giving the inmates their freedom, one by one, after each had been subjected to a sterilizing process or bath. The bath is also necessary on account of the bromidrosis common among the aged and which they do not perceive owing to the impairment of their olfactory organs.

A distinctive costume is as humiliating to the pauper as it is to the prisoner and it crushes self-respect more certainly than the prison stripes. Throughout this work stress has been laid upon psychic influence upon the organism and the sense of well-being of the aged. If we wish to improve the sense of well-being that conduces to happiness, we must avoid depressing influences and especially such that mortify and humiliate the aged. Such humiliation and the sense of inability to repair the cause, or attack the offender, destroy what little dignity and self-esteem the individual has left after accepting the bitter bread of charity. This soon leads to melancholia and dementia. The only advantage in having uniform costumes of a distinctive pattern is to make supervision simpler; possibly, too, there is a slight saving in the expense of clothing the inmates. Neither advantage is comparable to the advantage derived by the inmates from the knowledge that they are not obliged to wear the costume of the pauper.

In homes holding a large number of inmates, those having

marked mental deterioration should be removed from the others. As age advances many individuals become imitative like children and they are likely to imitate the actions and talk of the dullest of the inmates. Others become depressed when they are compelled to associate with dements, and may become likewise affected.

Many find in religion the consolation that makes them resigned to the inevitable. Aged women are especially amenable to religious influences and ministers of the Gospel find no more grateful subjects than the inmates of homes for aged women. Every opportunity should be given the inmates to worship according to their own faith and while it may not be practicable to have a separate chapel for each sect, where there is but one chapel it can be so fitted up that it will meet the requirements of the two great branches of Christianity, worshipping at different hours. Hebrews will not worship in a Christian chapel, but if there are many of that faith in a public non-sectarian institution any room can be converted into a temporary synagogue in a few minutes and at little expense. The head of such an institution, accustomed to handle all faiths and sects, will know the fast days and feast days of the Catholics, the Passover, the day of Atonement and other fast days of the Hebrews and other holidays kept by other faiths. The most important regulations to be observed on such days relate to food. If the head of such institution is ignorant of them, he can call to his aid either a well-informed inmate or a priest, minister or rabbi who will gladly advise him.

In all homes for the aged, except the alms-houses, there is a community of interest; it may be of religion or nationality or vocation, which binds all to a common object. There is also a sense of proprietary interest in the vocational, organization and private homes, which raises the inmates out of the class of paupers and dependents and entitles them to privileges and care which those in the alms-house have no right to demand. In the free homes maintained for certain nationalities and sects, the inmates are dependents but little better than paupers, and while the surroundings are far superior to the surroundings in the alms-house and the care is better, the inmates are still under the depressing influence of the sense of dependence upon the bounty of others. If we wish to increase the feeling of well-

being in the aged we must remove depressing influences. To the sensitive person the idea of being dependent upon charity is most humiliating, and if this idea is being constantly kept before the individual it will produce melancholia and it has led to suicide. For this reason let me repeat, the sight-seer and the professional sympathizer should be kept out of such institutions and the inmates should be allowed slight liberties, such as to go and come at will within certain hours, receive visitors, do work which will not interfere with the orderly conduct of the asylum, receive pay for such work and spend their earnings. At the same time rules relating to the introduction of unsuitable food and drink should be rigidly enforced even to the extent of expulsion of an inmate who brings in such articles surreptitiously.

Institutions of this kind often receive gifts of clothing. If worn clothing is received, it should be disinfected before distribution and the distribution should be made individually and in private, not as a public exhibition.

The vocational homes are generally homes of vocational organizations toward the maintenance of which the inmates have contributed, or else homes under government supervision for those who have been engaged in hazardous government occupations, and are offered as an inducement and prospective reward to those engaging in such work. A few vocational homes were founded by individual bequest and these are so well endowed that nothing is lacking to make the inmates contented and supply them with everything that can contribute to their welfare. The government asylums are mainly for soldiers and sailors, men accustomed to government routine and control and unaccustomed to home influences. These men can readily accommodate themselves to the new conditions and are not subjected to the mental depression associated with the idea of dependence upon charity. There is no such revolutionary change in their mode of life upon entering a government home as occurs in the life of the private individual who leaves his own home and family to enter an asylum. The vocational homes maintained by vocational organizations are like the homes maintained by fraternal organizations, private institutions to which the inmates have a certain proprietary right. Most organization asylums admit both sexes and thereby make institutional life more agreeable. To the old man or woman

accustomed to associate with the opposite sex, the sudden and complete deprivation of such association must produce a profound change in the mental attitude. If, in addition thereto, there is a change in the home surroundings and in the mode of living, the temperament of the individual becomes altered. This is a common observation in homes for the aged. The inmate soon after admission improves mentally and physically through the freedom from care, changed surroundings and a more regular mode of life. After a few weeks a temperamental change is noted and this depends upon the difference between the new mode of life and the life to which he had been accustomed. In a small house where the sexes mingle, as in the Actors' Fund Home on Staten Island, New York, the inmates form a large family. They occupy a cottage-like building which is to them a real home, not an asylum or an institution. They find here an approach to home conditions under probably more wholesome surroundings than formerly, with freedom from care about the future. This is an ideal home for the aged. The cottage plan of housing the old has received but little attention in this country, although the results obtained in the few small institutions occupying homelike cottages ought to commend it to those interested in the welfare of the aged. Where the cottage plan is impracticable, an effort should be made to copy home life as far as possible, by having small sleeping rooms instead of large dormitories, permitting aged couples to remain together, and fitting up their room with pictures and decorations from their old home, observing of course sanitary precautions. True, such an arrangement may give sleeping rooms a bizarre appearance and detract from the sense of order and neatness; it will, however, conduce to the happiness of the individual and may arouse worthy emulation and rivalry among the inmates. The object is, after all, to increase the happiness and prolong the lives of the aged persons by making them feel as much "at home" as is possible under institutional conditions and by preventing and relieving the little ailments which embitter and shorten their lives.

In all homes for the aged, music is the most acceptable and probably the most beneficial diversion. Even in the alms-house inmates will often be found who can play a musical instrument and who would gladly join an orchestra composed of inmates.

In such a nondescript orchestra it is not expected that the broken-winded trombone player will go through a Wagnerian opera or the tremulous fingers of the aged violinist will do justice to a nocturne. The aged prefer melody to harmony, and the old-time airs—which stimulate memory—to the airs of to-day. Not infrequently an old familiar air will rouse an individual from apathy and stimulate interest in life. To see an old pianist surrounded by a group of aged persons who are trying to sing in unison some sentimental song of a generation ago is a pathetic sight to the on-looker, but to the singers it means pleasure and happiness. In many other ways can the pleasures and happiness of the aged in institutions be enhanced. Men should be permitted to smoke in the open, but not in closed rooms; to play cards and other games, but not such as require much mental concentration or involve sudden exciting moments; harmless hobbies should not be interfered with, and little peccadilloes should be condoned. At no time in life does the vanity of women appear more silly than in old age; yet the vanity of the aged woman shown in an effort to appear younger and more charming is an indication of her interest in life. Instead of being condemned, this vanity or pride in appearance should be encouraged. The use of cosmetics does no harm nor does any harm result from her efforts to dress in the prevailing fashion. Flattery is as agreeable to the woman of seventy as to the girl of seventeen and is more beneficial. Care in dress and order in the room should be rigidly enforced, while increasing disorder in dress, appearance or surroundings should be looked upon as a gradual weakening of the emotions and of the mind as a whole.

The general lack of interest in geriatrics is responsible for the general neglect of the minor ailments of the aged. Some of these ailments have a pernicious psychic reaction, leading to delusions, which, with the increasing mental weakness, form senile paranoia. Presbyopia is generally neglected, little attention is paid to the teeth, virtually no attention is given to broken-down arches, or corns, bunions, and other pedal defects. The old man complains of pains and aches and they are set down as "rheumatic;" it is taken for granted that the old man will be constipated, and must urinate frequently; that the aged woman will have varicose veins and perhaps chronic ulcers on the legs,

intertrigo under the breasts, etc. Senile emphysema, senile tremor, senile debility, are dismissed with the remark "old age," and nothing more is done for the sufferer.

Senility is a state of physiological valetudinarianism. It requires special study, not as a pathological condition of maturity, but as an entity entirely apart from maturity and the person having charge of an institution for the aged should have the knowledge that comes from such study. This applies just as well to the physician who treats the ailments of the aged.

MEDICO-LEGAL RELATIONS

The most important and most frequent legal questions arising in connection with senility relate to the mental condition of the individual when making a will. It is recognized that old age carries with it mental impairment. Mental impairment is part of the organic and functional changes that constitute ageing, it is progressive, there are no remissions and it terminates in complete obliteration of the intellect. Long before this, the reasoning faculty and judgment become so impaired that the individual does not comprehend the nature of his acts, while memory is so weakened that he does not know the extent of his property or those who have natural claims upon him.

The mental functions and faculties do not become weakened uniformly and we consequently find some faculties stronger than others. Thus reason may be apparently as strong as in maturity yet memory and volition profoundly weakened. If there is a delusion, illusion or hallucination present, we have a form of insanity to deal with, a different proposition from the question of senile mental impairment.

It has been held that testamentary capacity is destroyed by actual weakness of the mind, "by anything that will weaken the individual's memory, judgment and volition in relation to the disposal of his property or the objects of his bounty." This might be made to include slight impairment, not sufficiently marked to attract the attention of the stranger who is not familiar with the normal mental condition of the individual. In another decision the judge declared, "the rule is that to avoid an instrument on the ground of the alleged mental incapacity of the person who executed the same, such person must have been so deprived of his mental faculties as to be wholly unable to understand or comprehend the nature of the transaction." Other decisions place other constructions upon the condition of mind necessary to indicate testamentary capacity. When memory has become so defective that the aged individual

does not know the extent of his property or the persons who have a natural claim upon it, or judgment is so weakened that he cannot intelligently dispose of it, his mental condition has passed beyond simple impairment; it is now senile dementia. There is no unanimity in either the medical or legal conception of the term "senile dementia." It has been applied to as slight an impairment as weakened memory alone. Some apply the term to that stage of impairment where the natural inhibition upon conduct is diminished or lost; some will not declare a case to be senile dementia until it has reached a stage where it becomes obvious to others who do not know the normal mental condition of the individual, while some authorities say mental derangement must accompany mental weakness. This last conception is wrong, for in many cases there is a gradual dulling of the faculties, an increasing difficulty in recalling events, in concentrating attention, in reasoning, in controlling the emotions, yet without mental perversion. If such an individual performs an irrational act it is through thoughtlessness, lack of reflection or impulse and not through illusions, delusions or hallucinations. In mental derangement the individual performs irrational acts because he thinks they are right. He is conscious that he has performed certain acts but his views concerning them are based upon false conception or belief or false perception with or without material basis and he will not accept rational views. The senile dement performs his acts without false conception or perception but rather unconsciously or impulsively and if any impression can be made upon his reasoning power if he can be sufficiently roused to realize that he has performed an act, he will recognize its sense or folly. It may be that owing to the frequent repetition of a story it may finally impress itself so vividly upon the mind as to produce therein the idea of reality and thus become a fixed delusion, but this in itself would not be a mental derangement. In old age the moral sense is frequently blunted and the lessened control over conduct may give rise to acts of immorality. In the popular conception of the term insanity these may be called insane acts, they are, however, not due to mental derangement but to mental and moral weakness. A distinction is to be made between mental derangement and mental impairment, although both may exist at the same time.

It has been held that forgetfulness of recent events is no evidence of incapacity to make a will. Forgetting the name of a member of the family does not imply such extensive mental impairment as to incapacitate the individual, but to forget the existence of a member of a family, especially if the person forgotten has not been absent sufficiently long to explain such forgetfulness, denotes profound mental weakness. It is always difficult to determine whether the omission from the provisions of a will of one having a natural claim upon the testator was intention or the result of defective memory. Occasional absent-mindedness due to mental concentration is no evidence of mental weakness, but persistent absent-mindedness or day dreaming, not due to mental concentration, shows profound mental impairment. To what extent this would affect the judgment of the individual while drawing up a will will depend upon his condition at the time. It is sometimes possible to arouse mental activity temporarily and maintain prolonged attention, under special stimulus, as when in danger or when facing some grave responsibility. Under such circumstances the individual would be in the same mental condition as during the lucid interval of paresis.

Judgment may be impaired in certain directions without affecting the disposing capacity, as where peculiarities and idiosyncrasies exist which do not impair the individual's memory or judgment as to the extent of his property and his obligations to his family. Hamilton says "the senile dement is prone to make foolish and trivial disposition of his property and particularly is this the case when he is aided by designing people who surround him, and the individual of this kind is very apt to be easily turned from his original purpose by fresh suggestions or new influences. He is liable to imposition and unjustifiable prejudice." The question of senile dementia depends upon the questioner's conception of the extent of the individual's mental impairment, evidenced by weakened memory, reasoning power and volition, as compared with the mental condition of the individual when at its best. In determining the extent of impairment it is necessary to compare all the faculties with the faculties as they were and not with the faculties of another of the same age. It is necessary to determine the form of dementia. The dementia of cerebral atrophy is progressive and

deepening while the post-apoplectic dementia is most marked immediately after the individual is roused after the attack, and gradually improves as the physical condition improves. The dementia of cerebral arteriosclerosis proceeds more slowly than the dementia of atrophy or softening and there are remissions. The dementia of cerebral softening is progressive and proceeds rapidly to complete obliteration of mentality. These would all be classed as senile dementia yet they differ in the extent and permanence of the mental impairment. A year after the recognition of senile dementia due to atrophy, the mind may still be sufficiently alert to understand and determine the provisions of the instrument that is being drawn. If it is a case of dementia due to softening a year after its inception (the embolic or thrombotic stroke) the mind is so profoundly impaired as to be incapable of comprehending the acts performed. A year after an apoplectic stroke the mind is comparatively clear. If a will is made a year after the appearance of the manifestations of dementia due to cerebral arteriosclerosis, it will be necessary to determine whether the will was made during a lucid interval or not. Not only is the dementia of arteriosclerosis profound but it is often associated with delusions or illusions. The terminal dementia which may occur in old age is the closing stage of other forms of insanity and needs no further comment, for there is no question about the testamentary incapacity when insanity has advanced to this stage. Various forms of insanity may appear in the aged but they are generally carried over from earlier life and are in an advanced stage when the individual reaches old age. Certain forms of mental derangement are peculiar to old age. Oikeiomania, an unreasonable hatred of one or more members of the family, is a rather frequent form of mental aberration. Beginning in a real or fancied slight or neglect on the part of some member of the family, the individual broods over it, dislike is aroused which develops into hatred, sometimes involving several members of the family. Later fears and persecutory delusions follow. The individual may be rational in every other direction but this one delusion impairs his judgment in one of the most vital points involved, namely, in making a will. (Contrary decisions have been rendered, however.) A will may be valid if an existing delusion does not interfere with the disposition of the prop-

erty. Delusions of grandeur are almost invariably associated with delusions of wealth, making the individual incapable of comprehending the real extent of his property. Where this exists the will shows internal evidence of mental derangement. The paranoic forms natural sequences and draws logical conclusions from a basic proposition which is an insane delusion. In his will he makes a rational disposition of his property, selects proper beneficiaries but he will dispose of vast sums he does not possess. A will made by a paretic during a lucid interval gives no evidence of insanity and unless other factors exist to invalidate it, it will be admitted. If made during an insane period it is incoherent, containing irrelevant comments, trivial bequests and other evidences of mental derangement. The disease, however, is rare in old age and when it occurs, the dementia proceeds rapidly, rendering the individual virtually incapable of making a will. As the mind becomes weakened, insane ideas become less extravagant, imagination is less active and there is mental confusion. Confusional insanity with and without delusions and hallucinations is occasionally found in the aged. In these cases mental concentration is impossible and the individual is incapable of making a coherent will.

What has been said of wills applies as well to other contracts and documents. The law does not recognize senile dementia other than as a form of insanity. The individual is either sane or insane, competent or incompetent, having sufficient mental capacity to make him responsible for his acts and make such acts represent his purpose and intent, or having not sufficient mental capacity to make him responsible for his acts and the acts valid. There is no border state in law, as there is in the medical aspect of the mental condition of insipient senile dementia and other conditions which we call the border state. The aged in the incipient stage of dementia frequently perform acts the nature of which they comprehend, but the consequences of which they cannot realize, owing to weakened mentality. Opposing views have been expressed as to the responsibility of the individual in this state and personal opinion, sentiment and public policy toward such acts, rather than the medical view of the mental condition, determine the nature of the act and the responsibility of the individual performing it. A factor heretofore unrecognized in determining the state of mind of

an aged person is the profound change in mentality during the senile climacteric. The senile climacteric marks the transitional period from old age to senility and while it is in progress, various forms of insanity may appear. This critical period lasts for several months and as there are many lucid intervals during this time, failure to recognize it will result in differences in the opinions of examiners who may see the individual passing through this stage, at different times. The mind is exceptionally clear during a lucid interval but if there has been considerable mental strain, as would occur when under prolonged examination, mental confusion appears and this is followed by delusions, maniacal outbursts, fits of depression, of crying or anger, confused or lost memory and perverted reason or judgment. There is no evidence of logical deduction, no rational or regular sequence in the character or order of these manifestations of insanity. Delusions are soon forgotten and new ones appear. There may be emotional exaltation at one moment, followed the next by calmness or depression. Memory is confused, recent and early events commingling and forming composite pictures which may give rise to delusions. During the senile climacteric there is sometimes a recrudescence of sexual desire which is not suppressed by reason or the sense of morals and this may give rise to sexual crimes. Oikiomania frequently develops during this time. Delusions of grandeur may arise but they are not the extravagant delusions found in paresis or paranoia, but rather exaggerated ideas of the indiivdual's importance or deeds. As the climacteric period gradually merges into the post-climacteric period or true senility, exaltation and depression give way to apathy, memory becomes more unstable, the reasoning power and judgment become weaker, delusions and other manifestations of mental derangement disappear and there is instead a progressive senile dementia. Undue influence is most frequently charged in senile cases and is based either upon temperamental supineness, intellectual impairment or weakened volition. The individual may be sane in every respect and it may be an evidence of his sanity that in order to be free from opposition, annoyance and worry he will leave to others the disposition of his property after his death. Under such circumstances the testator may make an error of judgment but there can be no question as to his intent or testamentary capacity. Cases

arise in which an aged individual, neglected by his family and cared for by strangers, makes the latter his beneficiaries. Such cases frequently lead to the charge of undue influence. If it can be shown that such strangers had poisoned his mind against his family, the inference is clear that they had designs upon the property and had used undue influence to obtain it. The case is different when the claimants are nephews or nieces or relatives still further removed, the testator had peculiarities which would have made him objectionable to them, and the persons taking care of him had done so in spite of the peculiarities and had given him the attention which made him comfortable during his life. Where undue influence is charged and intellectual impairment is shown, the decision will rest upon the extent of the mental impairment. The impariment may not be sufficiently marked as to destroy testamentary capacity, yet it may be sufficient to cause the individual to be easily turned from one purpose to another by suggestions of designing persons. Where weakened volition exists the individual will submit to insistent demands though made in the guise of suggestions. In these cases there is impaired judgment exhibited in the more or less complete dependence upon the judgment of others even in trivial matters. Where this condition exists a will drawn by the testator in favor of the person dominating him, and to the exclusion of others who have natural claims upon the testator, will be open to the charge of undue influence.

MARRIAGE

Most states have laws granting divorce or making marriage voidable or void on the grounds of insanity or physical incapacity at the time of marriage. One or the other of these causes is sometimes introduced to secure annullment of marriage with an aged person. The question of insanity generally hinges upon the construction placed upon senile dementia as a form of insanity and to what extent the physiological deterioration of the mind due to age has affected the reasoning faculties. This is discussed in the article on insanity.

Mesalliances occasionally involve legal questions respecting the mental capacity of an aged person contracting such marriage. In most cases such marriages are contracted under

sexual stress, the individual finding in marriage the only means of gratifying unimpaired sexual desires. In the absence of other evidences of insanity a mesalliance does not show mental deterioration.

Physical incapacity may be organic or functional, *i.e.*, due to defective organs or to weakness in the power of erection. The law demands that it be shown that the incapacity is incurable. In the coeundi impotentia senilis and in other forms of functional incapacity it is often possible to produce a temporary improvement under appropriate treatment, though in senile cases we cannot expect to maintain this improvement for any length of time. The weakness of the erectile power keeps pace with or may proceed faster than the gradual weakening of the organism and cure in the sense of complete restoration to the power of virile manhood is impossible. As the legal construction of the term "incurable," as applied to the annullment statutes of this state (New York) has not been decided, a nice question of law is raised whether the requirement of incurability will hold, notwithstanding possible temporary improvement. In other words does temporary improvement vitiate the sense of incurability. To the medical mind complete restoration of a normal function where that function was impaired or prevented implies a cure whether the restoration be temporary or permanent. Physical functional incapacity may be a relative impotence, the erectile power being impaired at certain times, under certain circumstances or in the presence of certain persons and not otherwise. This may occur in old age. From a medical standpoint it is impossible to declare functional incapacity incurable if the organs are normal.

SEXUAL PERVERSIONS

Sexual perversions are rather common in the aged and are due to either diminished power with undiminished desire or to a recrudescence of desire after power and desire had previously disappeared. The latter is generally a forerunner or early concomitant of senile dementia. The most prolific causes of sexual perversion in the young, depravity and inverted sexuality, are so rare in old age, that they can be practically eliminated. In the aged the causes are weakened mentality, dimin-

ished control over the emotions and some circumstance producing intense emotional excitement.

Hypererosis due to recrudescence of desire leads to impulsive acts like rape, especially upon children, and to bestiality. The offender does not realize the seriousness of his act nor does he seem to show much concern when caught in flagrante delicto. Close investigation into the mental condition of such offender will usually reveal mental defects involving the reasoning power, the emotions and the will. In almost every case there has been a clean record without any evidence of moral degeneracy until the commission of the crime. A peculiar feature of such offences is that the offender almost invariably selects a child about the age of puberty or younger, rarely an older one with whom the sexual act could be performed. These cases generally go to trial and unless the point is brought out that there is senile dementia, the offender is usually convicted. The following is a typical case.

An inmate of a soldiers' home who had not been away from the institution for several years visited a relative in the city. During the night he entered the room of a fourteen-year-old girl and attempted to assault her. The child ran screaming to her parents, who found the old man undressed in the child's room, although he had had plenty of time to get back to his own room during her absence. At the trial he denied all knowledge of being in the child's room and of the attempted assault. There was no mark of assault upon the child and the defense lay in discrediting the child's testimony.

He was discharged through disagreement of the jury. The following day he practised masturbation in a physician's office during the absence of the latter and did not desist when the physician returned. Although he gave evidences of failing memory and other mental impairment at the trial, his attorney failed to take advantage of this line of defense.

Outbursts of sexual fury during such recrudescence are liable to recur and for that reason where one has been guilty of an assault he should be kept under restraint.

Where the reasoning faculty is unimpaired the individual knows that assault is a heinous offence which is followed by punishment. Even during an outburst of sexual fury with diminished restraint upon the sexual instinct, this knowledge

saves him from committing the crime of rape, but it does not restrain him from bestiality and this is the usual form of sexual perversion occurring during the recrudescence of desire in the aged. This rarely comes to light. In most cases the individual is unable to perform the sexual act and adopts extraordinary measures to gratify his desire. In one case an aged manufacturer was found nude surrounded by a number of young women in like condition. When caught in the act he did not exhibit the slightest concern about his situation, yet he was able to conduct the affairs of a large factory and other commercial interests. Taken to a sanitarium he soon developed senile dementia with occasional sexual recrudescences. In another case a merchant was found by one of his family in a position depicted in a pornograph a number of which he had lying in front of him. The sight of these pictures aroused in him intense sexual excitement which he could not suppress or gratify. Thereafter the man was constantly guarded, he developed an oikeiomania (hatred of family), later obsession of persecution and senile paranoia.

The other class of cases of sexual perversion, those in which there is gradual loss of power with undiminished desire rarely lead to impulsive acts, as outbursts of sexual fury do not occur. These cases lead to solitary vice, occasionally to bestiality, more often to marriage with an unsuitable person.

In this class of cases there is no dementia and the individual, realizing his unfortunate condition, will often go to a physician for relief, invariably seeking restoration of power, never suppression of desire. The marital relations sometimes involve legal questions, but the other perversions are carried on so secretly that they are rarely discovered. Efforts to prove insanity by showing an unsuitable marriage have been made but unless there are other evidences of mental impairment, this alone is not sufficient to establish insanity.

MALINGERERS

Malingerers are frequently found among the aged, who either feign disease or exaggerate symptoms of an existing disease to create sympathy. The so-called factitious diseases which are produced voluntarily by the patient are rare among

the aged, nor do the aged purposely aggravate their ailments. They dread the infliction of pain and will do nothing which might give pain or increase it, or endanger their lives. As most of them have slight aches and pains, a little stiffness in their joints, a little difficulty of sight and hearing, they exaggerate their symptoms and in the weakened state of their minds the constant repetition and recital of their exaggerated symptoms may cause them to believe that they really suffer as much as they say. No one can measure the amount of pain or distress that a person feels, who has a disease which is ordinarily painful. The aged do not perceive pain as intensely as younger individuals, but they have a greater dread of it, the anticipation makes them more sensitive and if pain is inflicted their mental distress is greater. Under such circumstances a light tap is exaggerated into a blow, a mild breeze into a strong draught; they cry before they are hurt and claim to suffer all the pains that accompany the real injury. This is not real malingering as the patient has the mental impression of distress and for the same reason the hysteric should not be classed as a malingerer, for the pain is a mental reality though there be no visible cause. The true malingerer knows he tells an untruth for a purpose which will benefit him. Unconscious imitation may give rise to malingering. An aged man found a new companion who had a limp. He walked a short distance with his new friend every day and gradually fell into the step and limp of the other. His family called his attention to his unnatural walk and the old man at once concluded that he had some nervous disease. He exaggerated his symptoms, limped worse than ever before and when examined by the physician he insisted that he had a pain in the leg. The physician made note of the location of the pain and at his next visit read the record of the case as he had taken it but located the pain at another point of the leg.

The old man fell into the trap but held on to the limp. It was necessary to separate him from his friend, secure for him a new companion with sound legs and frighten him with threats of hospital and operation before he gave up the limp. The aged malingerer is rarely actuated by fear of punishment and he will not go to the length of those who will produce a disease or aggravate one, but he will maintain his deception even in the face of uncontrovertible proof. When he feigns a disease it is

invariably one in which pain is an element, never one which might cause his removal to an insane asylum or a hospital. The pain is referred to his chest or abdomen rarely to the back or other position of the body which he cannot see. It is worse when the person believes he is under obseration, but when his attention is diverted he gives no evidence of distress and under such circumstances pressure can be exerted on parts which a moment before could not stand the slightest touch. In examining a malingerer who feigns a disease, too close investigation and the appearance of doubt on the part of the physician will tend to fix in the malingerer's mind the symptoms he has given and make it more difficult to clear up the deception. In cases where deception is suspected the physician should have an assistant who without the knowledge of the person makes note of the spots where the patient says the pains are located. By directing attention to other portions of the body the malingerer will find other painful spots which should be noted in the assistant's record. The examination should be superficial without showing any doubt in the person's truthfulness. A second examination will usually bring out new painful points while the malingerer will have forgotten the location of the painful points of the former examination or remember only those to which his attention had been directed. In some cases the patient will present the appearance of decrepitude. This deception is difficult of detection by direct examination.

The person must be watched and caught unawares, or by arousing his emotions, either anger, joy or expectancy, a sufficiently powerful influence may be exerted to cause him to forget his assumed weakness. Threats of punishment, especially if the punishment involves pain, may cause him to betray himself. A few cases will be cited. A man aged seventy-four applied to the courts to compel one of his children to support him. He presented the appearance of extreme decrepitude, tottering along on his cane and requiring the assistance of a court attendent to help him to the witness chair. The case was decided against him and he was ordered out of the court room under threat of arrest. He went out without his cane and as spry as a young person. The aged mother of a prisoner appeared on the witness stand, her apparent weakness and scarcely audible voice arousing sympathy for her and her son.

When a later witness appeared against her son she abused him in a loud voice and it required some force to drag her from the court room. A woman aged seventy was receiving sick benefit from a society for total disability. She was apparently too feeble to walk and could go out only when assisted. A fire occurring in her house, she ran down two flights of stairs then remembering that she had forgotten something she ran back to her rooms and came out a second time alone.

The aged frequently exaggerate the symptoms of minor ailments and unless they give unusual symptoms it is almost impossible to detect the deception. Occasionally a single dose of some disagreeable drug will cure the patient or make the symptoms so mild that the dose need not be repeated. It is barbarous to apply a painful test to an aged person suspected of malingering, as his purpose is usually nothing else than to gain more sympathy and attention. There are occasions when the threat of a painful test is justifiable, as when an aged person complains that he is being neglected by his family in spite of their most solicitous care of him, and spreads the charge broadcast that they refuse to obtain for him medical attention, though there is nothing to indicate that he is ill except his complaints. Even here it is a question of humanity how far such threats should be carried out and how far the person should be humored. It is safe to say that when the patient will take disagreeable medicine and will submit to painful tests there is some basis for his complaints.

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